The Principles of Neurological Surgery

BY

LOYAL DAVIS, M.S., M.D., PH.D., D.SC., (HON.)

Professor of Surgery and Chairman of the Division of Surgery, Northwestern University Medical School, Chicago, Illinois

Fourth Edition, Thoroughly Revised,
With 186 Engravings, Containing 354 Illustrations
and 5 Plates, 4 in Color



LEA & FEBIGER

PHILADELPHIA

1953

Printed in the United States of America

PREFACE TO THE FOURTH EDITION

NEUROLOGICAL surgery has attained maturity as a surgical specialty in a comparatively short time. The first edition of this book was an attempt to make known to physicians and students the accomplishments possible in this field.

The wounded of World War II added a great deal to our knowledge of the traumatic aspects of neurological surgery by reason of the experiences of individual surgeons. It is to be hoped that organizational plans will be better laid to realize the utmost from combined and unified efforts in the event of a future catastrophe of such magnitude.

Again, I should say that this book does not include an exhaustive treatise upon each subject considered. I consider it unwise to include matters of surgical technique. If the student of medicine, regardless of age, is aided in obtaining a better concept of neurological surgery, the primary purpose will be achieved.

To the twelve men with whom I have been associated in their periods of training for neurological surgery, I wish to express my affection, respect and appreciation

Dr. Hale Haven	Dr. David Cleveland
Dr. John Martin	Dr. George Perret
Dr. Joseph Tarkington	Dr. Frank Padberg
Dr. Moses Ashkenazy	Dr. Stanton Goldstein
Dr. Daniel Ruge	Dr. Robert Anderson
Dr. Max Ramirez	Dr. Nicholas Wetzel

LOYAL DAVIS

CHICAGO

CONTENTS

CHAPTER I

Neurological Diagnosis

CHAPTER II

Craniocerebral Injuries

CHAPTER III

Intracranial Tumors

CHAPTER IV

Infection of the Central Nervous System

CHAPTER V

Infections and Tumors of the Skull

CHAPTER VI

Surgical Lesions of the Cranial Nerves

CHAPTER VII

Spinal Cord Injuries

CHAPTER VIII

Tumors of the Spinal Cord

CHAPTER IX

Injuries of the Peripheral Nerves

CHAPTER X

Surgical Treatments of Intractable Pain

CHAPTER XI

Surgery of the Autonomic Nervous System

CHAPTER XII

CHAPTER XIII

The Surgical Treatment of Intracranial Vascular Lesions

CHAPTER XIV

CHAPTER XV

CHAPTER XVI

Congenital and Acquired Anomalies of the Neural Axis

THE PRINCIPLES OF NEUROLOGICAL SURGERY

CHAPTER I

NEUROLOGICAL DIAGNOSIS

THE surgical treatment of diseases of the nervous system has reached the status of a mature surgical specialty. The teachings of a number of eminent neurologists, the wide sphere of experimental investigations and the contributions of many imaginative and ingenuous surgeons have made this possible in a relatively short period of time.

Many contributions have been made to the laboratory and surgical diagnosis of neurological diseases but collectively they cannot supplant the knowledge gained from physical and neurological examination.

Every doctor commonly encounters neurological cases in his practice, and the specialist in other diseases finds that many of his patients have neurological or neurosurgical complications. Invariably as a result of early teaching, the doctor may be confused, perplexed, bewildered and inarticulate when his patient presents symptoms of neurological significance. Often he concludes that the diagnosis of disorders of the nervous system is insurmountably difficult, that the prognosis is hopeless, and that treatment is hopeless.

This is not true provided one approaches the subject from the viewpoint of anatomy and physiology. If basic neuroanatomical and neurophysiological facts are applied, symptoms of diseases of the nervous system manifest themselves in a logical form. It should be remembered that it is impossible to memorize the signs, symptoms, and pathology of the various diseases of the nervous system without reference to those basic principles. The specialist must have a detailed knowledge of the nervous system, but the average physician need only remember a few of the long fiber tracts and some of their connections, the levels of reflexes, the levels of origin of cranial and spinal nerves, and the general form of the nervous system. These relatively simple, concise, practical facts may be readily acquired and mastered. They will provide an ample anatomical and physio-

a location would produce loss of cortical sensation over the opposite half of the body and blindness in the opposite half of the visual field in addition to paralysis of the opposite half of the body (*C*). Injury to the pyramidal fibers of the cerebral peduncles, pons, or upper part of the medulla produces a paralysis of the opposite half of the body and, in addition, may involve the corticobulbar fibers, the cranial nerve nuclei, or their emerging fibers. Involvement of the latter structures produces a paralysis of the muscles supplied by the cranial nerve upon the same side as the lesion, but the paralysis of the body is on the opposite side (*D* and *E*). While damage to the pyramidal tracts within the spinal cord may be unilateral (*F*), it is common for both sides to be injured (*G*).

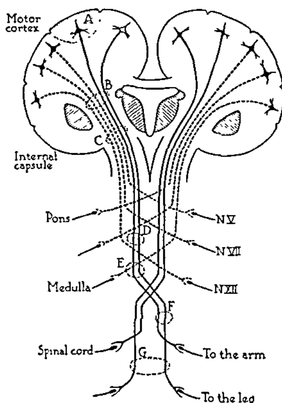


FIG. 2.—Diagram of the possible location of lesions which may affect the upper motor neuron.

In the *flaccid* type of paralysis, smaller groups of muscles are affected, and there is a loss of muscle tone, absent deep tendon reflexes, muscular atrophy, and the muscles do not respond to the faradic current while upon galvanic stimulation, their reaction is slow and serpentine. These symptoms are produced by a lesion of the *lower motor neuron*.

Lower motor axons arise from the cells of the anterior gray column of the spinal cord and from the cells of the motor nuclei of the cranial nerves. The fibers from the spinal cord emerge through the anterior spinal roots and end in the skeletal muscles, while those from the brain stem are contained in the cranial nerves and end in the striated muscles.

Lesions of the *lower motor neuron* occur in a very few locations. These are the anterior motor horn cells or their emerging fibers in the anterior roots, the nerve plexuses, the peripheral nerves, and the cauda equina of the spinal cord.[1]

There are a few practical points in making a neurological examination which will allow the examiner to detect the disorders of muscular function more easily and with more interest.

When gross weakness occurs, the examination is a relatively simple matter, but often the patient is able to perform all movements apparently with equal strength. Pollock has emphasized the importance of observing the apparently trivial signs which are present when gross methods of examination fail to reveal any motor disturbance.

As one observes a patient lying upon his back, the palpebral fissure may be wider, and spontaneous winking less frequent or absent on the affected side. The corneal reflex may not be elicited as effectually. The upper eyelid upon the paretic side may be lifted upward by the examiner more easily than upon the unaffected side; and whereas, the normal lid quickly resumes its normal position, the affected lid slowly and incompletely covers the eyeball. The difference in muscle tone in the upper eyelids can be seen and felt very definitely. A voluntary attempt to close the eyelids forcibly will produce greater wrinkling of the skin about the lids on the unaffected side.

The aperture of the naris on the paretic side is smaller, and the nasolabial fold is not as deep. When the patient shows his teeth, the affected side may lag slightly or the nasolabial fold may not increase in depth as it does on the normal side. A strong attempt to show the teeth is always accompanied by a strong contraction of the platysma muscle on the normal side, but this is often absent on the weak side. The facial movements should be observed carefully upon voluntary contraction and in response to emotional expression. Often no weakness can be observed when the patient's face is in repose or when the facial muscles are contracted voluntarily, and yet when he talks, cries, or laughs, the difference in strength is easily detected.

In the absence of a demonstrable weakness of an upper extremity, some clumsiness, defect in associated movements, greater fatigability, or tremor will be found on the affected side. With the patient lying on his back and with both of his upper extremities outstretched before him, the affected extremity will fall downward slightly or

[1] There are, in addition, paralyses which are due neither to an upper nor lower motor neuron lesion. These are the recurrent and transient palsies, such as those which occur in myasthenia gravis, intentional hypertonia, family periodic paralysis and catapley These disturbances are of greater interest to the neurologist.

rotated passively, it may strike the face in its precipitous descent. In contrast, the unaffected extremity falls slowly and rarely, if ever, strikes the face. Often the strong arm remains extended for a time before it descends. Likewise, when the affected lower extremity is passively raised and then dropped, it falls rapidly to the bed. If flexed passively with the heel resting on the bed and then released suddenly, the extremity quickly falls to an extended position in outward rotation. (Fig. 4.) A lower extremity with normal tone, so handled, gradually returns to its original position slowly and with

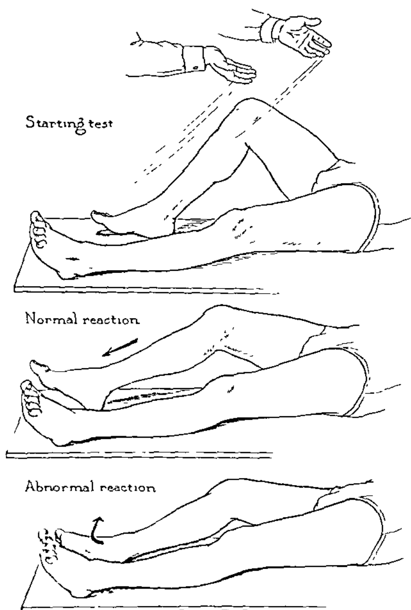


FIG 4 – Method for testing the relative strength of the lower extremities in a stuporous or comatose patient

distinct evidence of the presence of a check mechanism at work. Although both upper and lower extremities may react by flexor withdrawal to stimulation with a pin prick, the character of the movement often differs in degree or effectiveness.

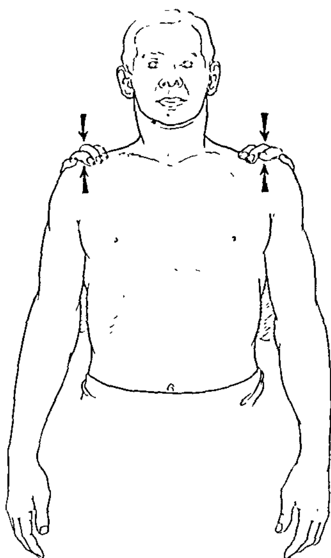


FIG. 5.—The strength of the trapezius muscles may be tested by having the patient shrug his shoulders against resistance.

A similar examination may be employed to determine weakness or paralysis of the muscles supplied by the cranial nerves. The nasolabial fold is deeper on the normal side, and if drooling is present, it occurs at the angle of the paretic side of the lips. With expiration the paretic cheek often puffs out while upon inspiration the aperture of the naris on the unaffected side becomes wider. When an attempt is made to separate the eyelids, resistance on the normal side is greater. On the paretic side the eyeball often rolls upward when the attempt to resist the pressure is ineffectual.

While these tests will make it easier to detect weakness in an extremity, it is often necessary to examine the strength of individual muscles or muscle groups. This is particularly true in the presence of lesions of the spinal cord or peripheral nerves.

The strength of the *trapezius* muscle may be tested by having the patient shrug his shoulders against resistance. If the examiner stands in front of the patient while he is performing the test, con-

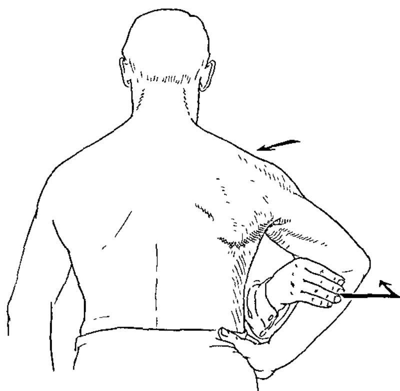


FIG. 6 —The rhomboid muscles can be tested by having the patient place his hand on his hip and then bring his shoulder backward while the examiner resists this movement by pushing the elbow forward.

traction of the *levator scapulæ* muscle may be palpated. This muscle is innervated by spinal nerves which originate from the third, fourth and fifth spinal cord segments. By repeating the test and standing behind the patient, contraction of the upper portion of the trapezius muscle, which is mainly responsible for shrugging the shoulders, can be tested. The upper portion of the trapezius is innervated by the eleventh cranial nerve (accessory). The trapezius is also the principle muscle used in keeping the shoulder braced Contraction of the middle and lower portions of the muscle can be noted along the vertebral margin of the scapula when the shoulders are moved back and adducted. If the examiner resists this movement by bringing

the outer portion of the patient's shoulder forward, contraction of the trapezius can be seen and felt. (Fig. 5.)

The *rhomboid* muscles can be tested by having the patient place his hand on his hip and then bring his shoulder backward while the examiner resists this movement by pushing the elbow forward. These muscles are innervated by the dorsal scapular nerve which arises from the fourth and fifth cervical segments of the spinal cord. (Fig. 6.)

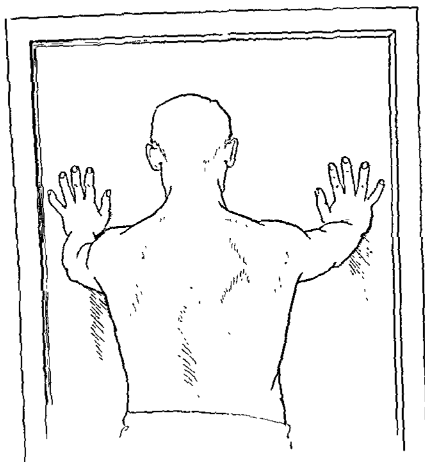


FIG. 7. When a patient pushes forward firmly against resistance the vertebral borders of the scapulæ are kept closely applied to the thoracic wall by the serratus anterior muscles. When they are paralyzed, "winging" of the *scapula occurs.*

Normally when a patient pushes firmly against resistance, the vertebral borders of the scapulæ remain closely applied to the thoracic wall. The *serratus anterior* muscle keeps the scapula in this position and when it is weak or paralyzed a characteristic "winging" of the vertebral border of the scapula occurs. The long thoracic nerve which arises from the fifth, sixth and seventh cervical segments of the spinal cord innervates the serratus anterior. (Fig. 7.)

Medial or internal rotation of the arm at the shoulder is performed chiefly by the *subscapular* and *teres major* muscles. To test the strength of the muscles the forearm should be flexed at a right angle, which serves as a lever for the examiner to resist the movement by attempting to externally rotate the arm. To test the strength of external or lateral rotation of the arm the above test should be reversed, and the examiner should stand behind the patient to see and feel contraction of the *infraspinatus* muscle which is the prime muscle involved and is innervated by the suprascapular nerve originating from the C5 and 6 cervical segments of the spinal cord. (Fig. 8.)

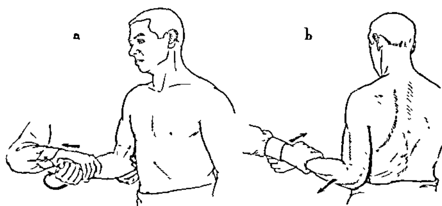


FIG. 8 —To test the strength of the subscapular and teres major muscles, the examiner should resist internal rotation of the arm at the shoulder (*a*) and to test the strength of the infraspinatus muscle, which is the prime external rotator of the arm, the test should be reversed (*b*).

If the patient attempts to abduct the arm from the resting position at the side against resistance, another method is provided for testing the strength of the *supraspinatus* and *trapezius* muscles. If the patient abducts the arm from the side to an angle of 60 degrees and then attempts to hold it in that position against the examiner's resistance, the strength of contraction of the *deltoid* muscle can be seen and palpated. This muscle is innervated by the axillary nerve from the cervical fifth and sixth segments of the spinal cord. (Fig. 9.)

When the patient abducts his arm to an angle of 90 degrees and attempts to hold it there against resistance, the deltoid and supraspinatus muscles are chiefly concerned although the infraspinatus, teres minor and subscapularis muscles which externally rotate the scapula also take part in the movement. It should be remembered that elevation of the arms at the shoulder joint may be accomplished by abduction or by forward flexion. The latter movement requires contraction of the pectoralis major muscle while elevation in abduc-

tion does not. The external, or lateral, rotators of the scapula play a part in elevating the arm to the horizontal position, but they do not come into full participation until elevation passes that level. During the entire movement of the arm, the serratus anterior and trapezius muscles fix the scapula to the chest wall and when the arm is raised above the horizontal, the trapezius aids in elevation of the scapula.

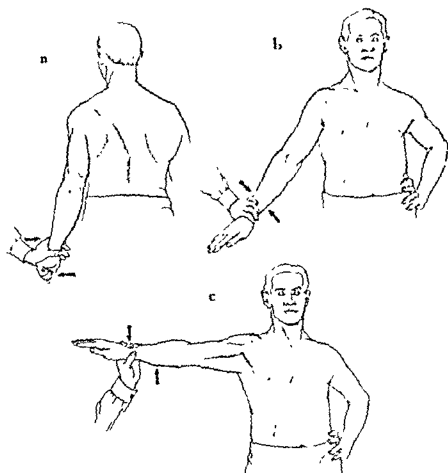


Fig 9 —(*a*) Resistance to abduction of the arm from the resting position at the side tests the strength of the supraspinatus and trapezius muscles, (*b*) abduction of the arm from the side to an angle of 60 degrees against resistance tests the strength of the deltoid muscle, (*c*) abduction of the arm to an angle of 90 degrees against resistance tests the strength of the deltoid and supraspinatus muscles primarily. The infraspinatus, teres minor, and subscapularis muscles which externally rotate the scapula also take part in the movement.

The strength of the *pectoralis major*, *teres major* and *latissimus dorsi* muscles can be tested by having the patient adduct the horizontally abducted arm against resistance The *sternocostal* and clavicular portions of the pectoralis major can be palpated and seen to contract when the patient attempts to bring his elbow forward against resistance. Or, the arm may be first abducted and slightly

flexed at the elbow and then the patient asked to retain that position while the examiner attempts to abduct the arm further. Contraction of the pectoralis major and latissimus dorsi muscles can be palpated quite easily. The teres major muscle is innervated by the lower subscapular nerve (C5 and C6), the latissimus dorsi by the thoracodorsal nerve (C6, C7 and C8), the sternocostal portion of the pectoralis major by the medial anterior thoracic nerves (C5, 6, 7, 8 and Th. 1) and the clavicular portion by the lateral anterior thoracic nerves from the same spinal cord segments. (Fig. 10.)

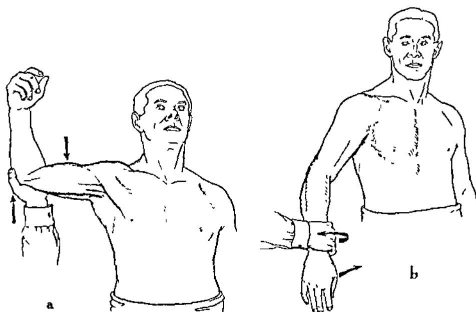


FIG 10 —(*a*) The strength of the pectoralis major, teres major, and latissimus dorsi muscles can be tested by having the patient attempt to adduct the horizontally abducted arm against resistance, or (*b*) the arm may be first abducted and slightly flexed at the elbow and the patient asked to retain that position while the examiner attempts to abduct the arm further

The *biceps* and *brachialis* muscles can be tested by having the patient flex the completely supinated forearm and resist an attempt by the examiner to straighten the arm. During this movement the contracting biceps can be seen and palpated. Both of these muscles are innervated by the musculocutaneous nerve which originates from the fifth and sixth cervical spinal cord segments. If the forearm is placed in a position midway between pronation and supination and the forearm is flexed against resistance, the belly of the *brachioradialis* muscle contracts strongly and this muscle is innervated by the fifth and sixth cervical segmental components of the radial nerve. Even when the biceps and brachialis muscles are completely paralyzed the brachioradialis is capable of strongly flexing the forearm providing it is in pronation. (Fig. 11)

If the patient attempts to extend the slightly flexed forearm against resistance, contraction of the *triceps* muscle can be palpated and observed. Care must be taken to so place the arm that the effect of gravity may be eliminated. The triceps muscle is innervated by the radial nerve (C7, 8 and Th. 1). (Fig. 12.)

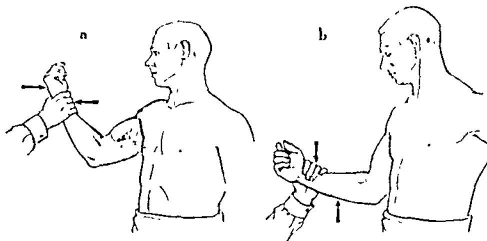


FIG. 11.—(*a*) Testing the biceps and brachialis muscles by having the patient flex the completely supinated forearm against resistance; (*b*) with the forearm midway between pronation and supination the forearm is flexed by the brachioradialis.

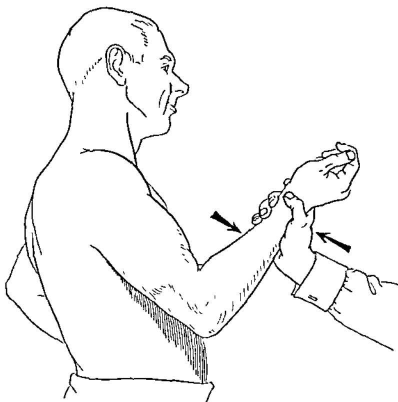


FIG. 12.—Contraction of the triceps muscle can be palpated and observed when the patient attempts to extend the slightly flexed forearm against resistance.

Supination of the forearm depends primarily upon the *biceps* and the *supinator* muscles which are innervated respectively by the musculocutaneous and radial nerves; pronation is performed mainly by the *pronator teres* and *pronator quadratus* muscles, both of which are innervated by the median nerve. The strength of these muscles can be tested by flexing the forearm to a right angle, slightly rotating the shoulder externally and keeping the elbow in contact with the body while the patient attempts to supinate while the examiner tries to pronate the forearm and *vice versa*. (Fig. 13.)

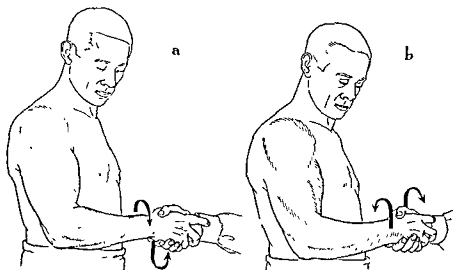


FIG. 13.—(*a*) Supination of the forearm depends upon the biceps and supinator muscles; (*b*) pronation is performed by the pronator teres and pronator quadratus muscles The strength of these muscles can be tested by flexing the forearm to a right angle, slightly rotating the shoulder externally and keeping the elbow in contact with the body while the patient attempts to supinate and the examiner attempts to pronate and *vice versa*.

With the hand resting flat on the top of a table, the power of the extensors of the hand at the wrist may be tested and each of the muscles participating in the action should be palpated separately. The *extensor carpi ulnaris* (C7, 8), the *extensor carpi radialis longus* (C6, 7) and the *extensor digitorum* (C7, 8) are all innervated by the radial nerve. (Fig 14.)

Flexion of the wrist is performed chiefly by the *flexor carpi ulnaris* and the *flexor carpi radialis*. (Fig. 15, *a*.) If the examiner offers resistance to flexion of the little finger, the tightened tendon of the flexor carpi ulnaris can be palpated. Or, if the examiner presses on the palmar surface of the outstretched little finger and resists attempts of the patient to abduct it, the same phenomenon may be observed If attempts of the patient to flex the hand at the wrist

against resistance, placed against the fingers, the tendons of the flexor carpi radialis, and to a less degree, the palmaris longus, can be seen to contract strongly. The flexor carpi ulnaris is innervated by the ulnar nerve (C7 and C8) and the flexor carpi radialis by the median nerve (C7 and C8).

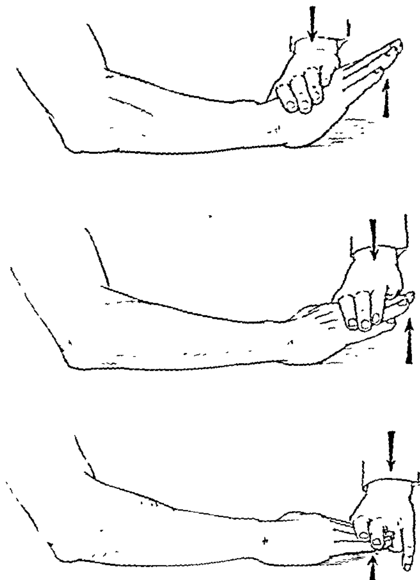


FIG. 14.—Extension of the hand at the wrist is dependent upon contraction of the extensor carpi radialis longus, the extensor carpi ulnaris, and the extensor digitorum.

The thumb may be adducted in the plane of the palm (ulnar adduction) or at right angles to the plane of the palm (palmar adduction). In the first movement a number of muscles take part but the prime mover is the *adductor pollicis*, innervated by the ulnar nerve (C8 and Th. 1). (Fig. 17,*a*.) In palmar adduction of the thumb the dorsal and palmar muscles of the first interosseus space are tested by attempting to withdraw a piece of paper which the

Supination of the forearm depends primarily upon the *biceps* and the *supinator* muscles which are innervated respectively by the musculocutaneous and radial nerves; pronation is performed mainly by the *pronator teres* and *pronator quadratus* muscles, both of which are innervated by the median nerve. The strength of these muscles can be tested by flexing the forearm to a right angle, slightly rotating the shoulder externally and keeping the elbow in contact with the body while the patient attempts to supinate while the examiner tries to pronate the forearm and *vice versa*. (Fig. 13.)

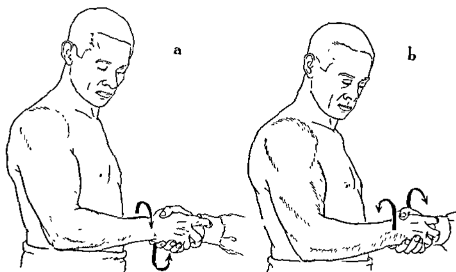


FIG 13.—(*a*) Supination of the forearm depends upon the biceps and supinator muscles, (*b*) pronation is performed by the pronator teres and pronator quadratus muscles. The strength of these muscles can be tested by flexing the forearm to a right angle, slightly rotating the shoulder externally and keeping the elbow in contact with the body while the patient attempts to supinate and the examiner attempts to pronate and *vice versa*.

With the hand resting flat on the top of a table, the power of the extensors of the hand at the wrist may be tested and each of the muscles participating in the action should be palpated separately. The *extensor carpi ulnaris* (C7, 8), the *extensor carpi radialis longus* (C6, 7) and the *extensor digitorum* (C7, 8) are all innervated by the radial nerve. (Fig. 14.)

Flexion of the wrist is performed chiefly by the *flexor carpi ulnaris* and the *flexor carpi radialis*. (Fig. 15, *a*.) If the examiner offers resistance to flexion of the little finger, the tightened tendon of the flexor carpi ulnaris can be palpated. Or, if the examiner presses on the palmar surface of the outstretched little finger and resists attempts of the patient to abduct it, the same phenomenon may be observed. If attempts of the patient to flex the hand at the wrist

against resistance, placed against the fingers, the tendons of the flexor carpi radialis, and to a less degree, the palmaris longus, can be seen to contract strongly. The flexor carpi ulnaris is innervated by the ulnar nerve (C7 and C8) and the flexor carpi radialis by the median nerve (C7 and C8).

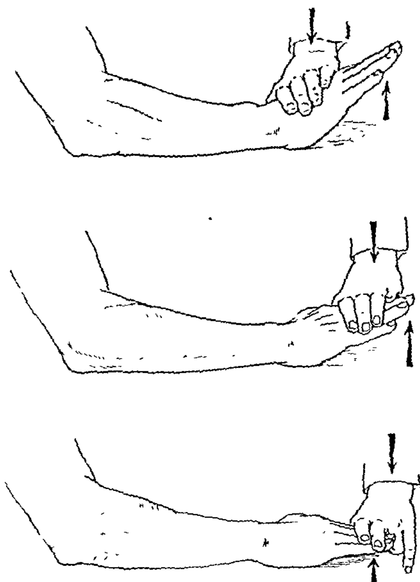


Fig. 14 —Extension of the hand at the wrist is dependent upon contraction of the extensor carpi radialis longus, the extensor carpi ulnaris, and the extensor digitorum.

The thumb may be adducted in the plane of the palm (ulnar adduction) or at right angles to the plane of the palm (palmar adduction). In the first movement a number of muscles take part but the prime mover is the *adductor pollicis*, innervated by the ulnar nerve (C8 and Th. 1). (Fig. 17,*a*.) In palmar adduction of the thumb the dorsal and palmar muscles of the first interosseus space are tested by attempting to withdraw a piece of paper which the

proximal phalanx and in the second movement the metacarpal bone. With the thumb in the position of palmar adduction to obviate the action of other muscles and with the proximal phalanx immobilized by the examiner flexion of the distal phalanx of the thumb which is performed by the *flexor pollicis longus* muscle can be tested. This muscle is innervated by the median nerve (C7, 8 and Th. 1). (Fig. 16, *b*.)

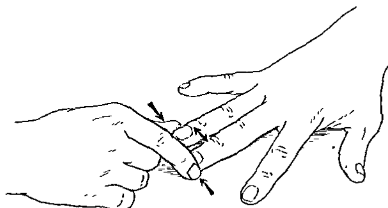


Fig. 18.—Abduction and adduction of the fingers is performed by the dorsal and palmar interossei muscles respectively.

Opposition of the thumb and the little finger may be tested by placing a piece of paper between the tips of the patient's thumb and little finger and the examiner attempting to pull it away. When both the thumb and little finger are extended their tips meet and the two digits form a vertical arch over the palm. In this movement the thumb is drawn over the palm chiefly by the *opponens pollicis* muscle which is innervated by the median nerve (C8 and Th. 1). Opposition of the little finger is accomplished by palmar elevation of the fifth metacarpal as well as by action of the opponens muscle (Ulnar nerve, C8 and Th. 1). (Fig. 17, *b, c*.)

Abduction and adduction of the fingers is performed by the *dorsal* and *palmar interossei* respectively, which receive their nerve supply from the ulnar nerve (C8 and Th. 1). The patient may be required to hold a piece of paper between adjacent fingers against efforts to remove it to test the power of the adductors. With the fingers fully extended and spread apart flat on a table, the patient resists the examiner's attempt to bring the fingers together. This act tests the strength of the dorsal interossei muscles. (Fig. 18.)

Extension of the fingers at the metacarpophalangeal joints is accomplished by the *extensor digitorum* muscle, aided by the extensor muscles of the index and little fingers, all of which are innervated by

the radial nerve (C7 and C8). Extension at the metacarpophalangeal and interphalangeal joints must be tested separately. To test the power of extension at the proximal interphalangeal joint of the index finger, for example, the metacarpophalangeal joint should be extended and the proximal interphalangeal joint partly flexed. The patient should then attempt to extend the second phalanx against resistance. This tests the strength of the *first lumbrical* and *interosseous* muscles which are supplied respectively by the median and ulnar nerves. Full extension at all interphalangeal joints is performed by the lumbricals and interossei with the help of the extensor digitorum. The interossei and the fourth and third lumbricals are innervated by the ulnar nerve (C8 and Th. 1) and the second and first lumbricals by the median nerve (C7 and C8).

Flexion of the distal phalanges of the fingers is brought about by contraction of the *flexor digitorum profundus*. (Fig. 15, *c*.) The portions of this muscle which go to the index and middle fingers are innervated by the median nerve and those which go to the ring and little fingers are supplied by the ulnar nerve. In both instances the spinal cord segments represented are C7, 8 and Th. 1. Flexion of the middle phalanges of all the fingers is performed by the *flexor digitorum sublimis* which is innervated completely by the median nerve from the same segments, though principally C8 and Th. 1. (Fig. 15, *b*.) Unless the distal phalanges are completely relaxed the flexor digitorum profundus may take part in this movement. Flexion of the fingers at the metacarpophalangeal joints with extension of the phalanges is performed by combined action of the *palmar* and *dorsal interossei* and to a lesser degree, the *lumbricals*. The extensor digitorum muscle aids in extension of the phalanges. The interossei are innervated by the ulnar as are the third and fourth lumbricals; the first and second lumbricals by the median. Flexion of the thumb is performed at the phalangeal joint by the *flexor pollicis longus* and at the metacarpophalangeal joint by the *flexor pollicis longus* and *brevis* as well as by the *abductor pollicis brevis*. All of these movements described are those employed in order when a patient is asked to close his fist but in addition the thumb is extended before it is flexed and brought across the palm. Since a tight firm fist can be made only with the wrist in extension, the extensor carpi ulnaris and the radial extensors of the wrist play a synergistic rôle in this movement.

The abdominal muscles may be observed to contract and palpated if the patient is asked to flex his head against resistance when he is lying on his back. During this movement attention should be paid to movement of the umbilicus upwards or downwards, depending upon weakness of the upper and lower halves of the *rectus*

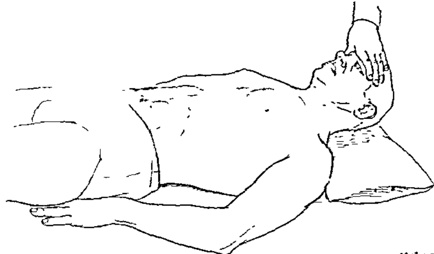


FIG 19.—The rectus abdominis muscles may be observed to contract if the patient flexes his head against resistance when he is lying on his back.

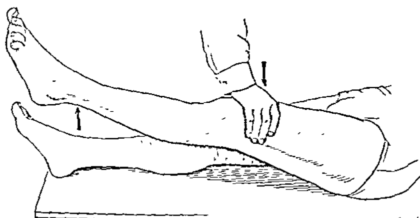


FIG. 20 —Flexion of the hip may be tested by having the patient raise the fully extended leg off the bed against resistance. This movement is chiefly dependent upon the iliopsoas muscle.

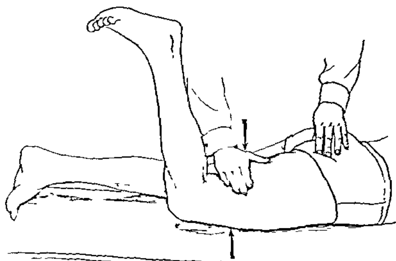


FIG 21 Extension at the hip may be tested when the patient lies prone and lifts his leg flexed at the knee, off the bed and holds it there against the examiner's attempts to force it back to the bed Contraction of the gluteus maximus muscle can be seen and felt.

abdominis and the *external* and *internal oblique* muscles. These muscles are innervated by the lower thoracic spinal nerves originating from Th. 6 to L 1 segments of the spinal cord. The umbilicus generally is regarded as at the level of Th. 10. (Fig. 19.)

Flexion of the hip may be tested by having the patient raise the fully extended leg off the bed against resistance. (Fig. 20.) This movement is chiefly dependent upon the *iliopsoas* muscle which is supplied by the femoral nerve (L2, 3 and 4). Extension at the hip may be tested when the patient lies prone and lifts his leg, flexed at the knee, off the bed and holds it there against the examiner's attempts to force it back to the bed. The contraction of the *gluteus maximus* (L5, S1 and 2) which is responsible for the act can be seen and felt. (Fig. 21.)

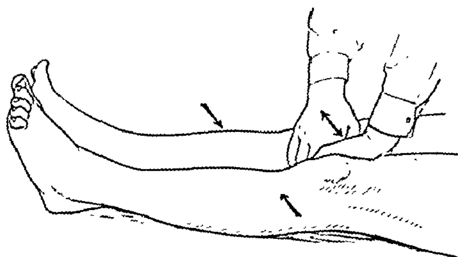


Fig 22.—The power of adduction of the legs can be tested by the examiner attempting to force the legs apart against the patient's efforts to keep them close together. The adductor muscles tested are the adductor magnus, adductor brevis, adductor longus, lower fibers of the gluteus maximus, and the quadratus femoris.

The power of adduction of the legs can be tested by the examiner attempting to force the legs apart against the patient's efforts to keep them close together. (Fig. 22.) The adductors of each leg may then be tested separately and contraction of the muscle group palpated directly. The adductor muscles tested are the *adductor magnus* (the most powerful), *adductor brevis*, *adductor longus*, lower fibers of the *gluteus maximus* and the *quadratus femoris*. The adductor magnus is innervated by the obturator nerve (L3 and 4) and the sciatic nerve (L4 and 5) This test may be reversed to test the power of abduction of the thigh, that is, the patient attempts to abduct his thigh against resistance. The *gluteus medius* muscle is the most powerful abductor of the thigh, and it is supplied by the superior gluteal nerve (L4 and 5).

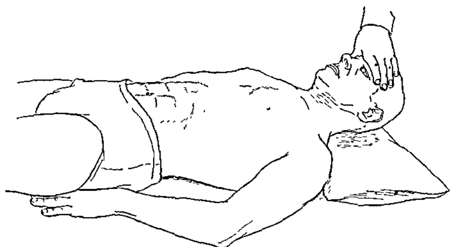


FIG 19.—The rectus abdominis muscles may be observed to contract if the patient flexes his head against resistance when he is lying on his back.

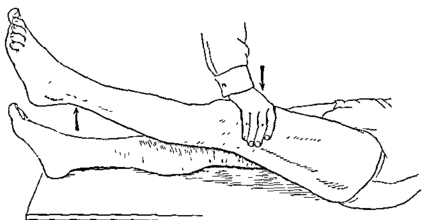


FIG. 20.—Flexion of the hip may be tested by having the patient raise the fully extended leg off the bed against resistance. This movement is chiefly dependent upon the iliopsoas muscle.

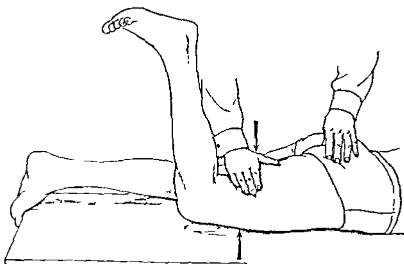


FIG 21.—Extension at the hip may be tested when the patient lies prone and lifts his leg, flexed at the knee, off the bed and holds it there against the examiner's attempts to force it back to the bed. Contraction of the gluteus maximus muscle can be seen and felt.

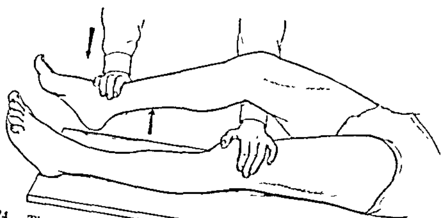
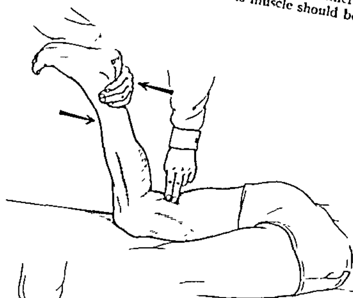


FIG 24 — The power of extension at the knee may be tested by having the patient attempt to extend the leg at the knee against the examiner's efforts to flex the knee. Contraction of the quadriceps femoris muscle should be observed and palpated.



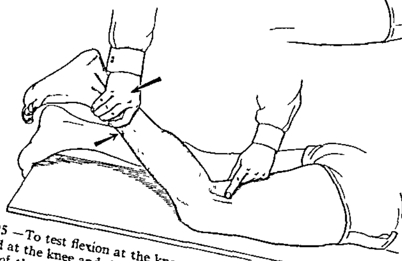


FIG 25 — To test flexion at the knee the patient should lie prone with the lower leg flexed at the knee and the examiner should pull the foot toward the bed Contraction of the semitendinosus and semimembranosus muscles on the medial side of the popliteal space and the biceps femoris muscle situated laterally should be observed and palpated

Medial and lateral rotation of the leg at the hip may be tested by *having the patient* lie prone with the lower leg flexed at the knee. He attempts to move the lower leg laterally while the examiner tries to pull it medially. The reversal of these actions tests the strength

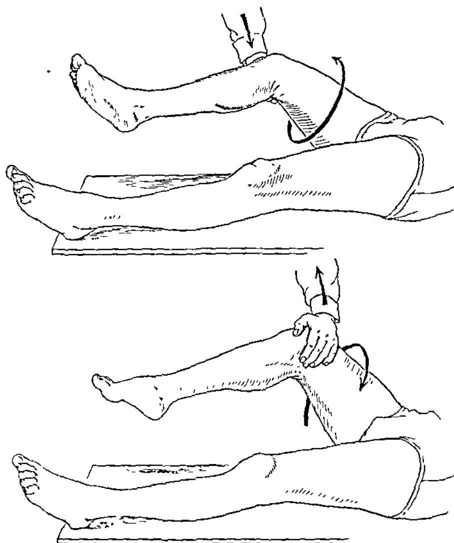


FIG 23.—Medial and lateral rotation of the leg at the hip may be tested by having the patient lie prone with the lower leg flexed at the knee. He attempts to move the lower leg laterally while the examiner tries to pull it medially. The reversal of these actions tests the strength of the medial rotators of the leg.

of the medial rotators of the leg. Medial rotation is performed by the *gluteus medius* muscle which can be palpated when it contracts. Lateral rotation at the hip is performed by the action of a number of muscles, the most important of which is the *gluteus maximus*, innervated by the inferior gluteal nerve (L5 and S1, 2). (Fig. 23.)

The power of extension at the knee may be tested by having the patient attempt to extend the leg at the knee against the examiner's

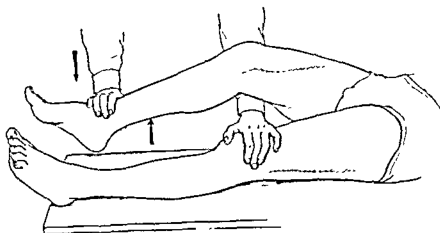


FIG. 24.—The power of extension at the knee may be tested by having the patient attempt to extend the leg at the knee against the examiner's efforts to flex the knee. Contraction of the quadriceps femoris muscle should be observed and palpated.

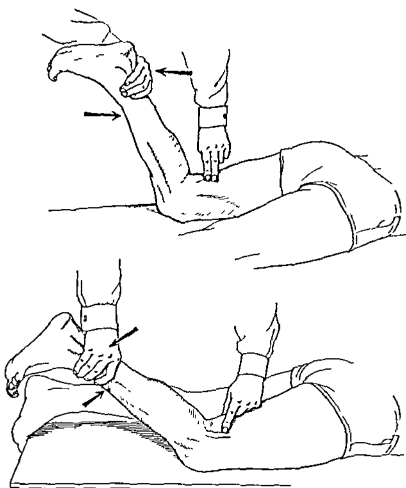


FIG. 25.—To test flexion at the knee the patient should lie prone with the lower leg flexed at the knee and the examiner should pull the foot toward the bed. Contraction of the semitendinosus and semimembranosus muscles on the medial side of the popliteal space and the biceps femoris muscle situated laterally should be observed and palpated.

efforts to flex the knee. Contraction of the *quadriceps femoris* muscle should be observed and palpated. The examiner may use his arm beneath the knee to act as a fulcrum in testing this action. The femoral nerve (L2, 3 and 4) innervates the quadriceps. (Fig. 24.) To test flexion at the knee accurately the patient should lie prone with the lower leg flexed at the knee and the examiner should pull the foot toward the bed. Contraction of the *semitendinosus* and *semimembranosus* muscles on the medial side of the popliteal space and the *biceps femoris* muscle situated laterally should be observed and felt. These muscles are innervated by the sciatic nerve (L5, S1 and 2). (Fig. 25.)

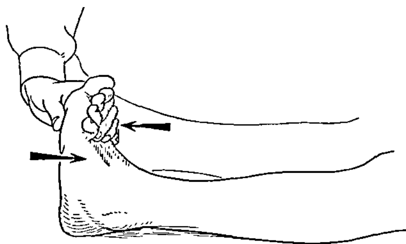


FIG. 26.—If the patient is asked to dorsiflex his foot against resistance, the tibialis anterior and extensor digitorum longus muscles may be seen to contract strongly.

If the patient is asked to dorsiflex his foot against resistance, the *tibialis anterior* and *extensor digitorum longus* muscles may be seen to contract strongly. The latter muscle can be tested further by the examiner's attempts to depress the patient's dorsiflexed toes. (Fig. 26.) Both muscles are supplied by the peroneal nerve (L4, 5 and S1). Plantar flexion of the foot is performed mainly by the *gastrocnemius* muscle and it should be palpated during attempts of the patient to flex the foot against resistance. The soleus, tibialis posterior, flexor digitorum longus, and flexor hallucis longus also take part in this movement. The gastrocnemius is innervated by the tibial nerve (L5, S1, 2 and 3). (Fig 27.)

To test the patient's power to invert the foot, he should turn it into that position and resist the examiner's attempts to evert the foot. The opposite actions should be employed to test the patient's power of eversion of the foot Inversion is produced by the action

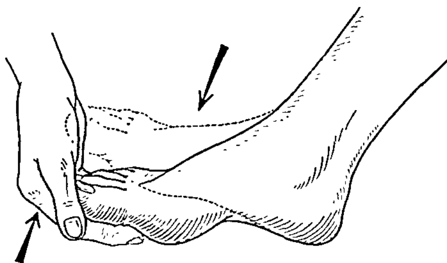
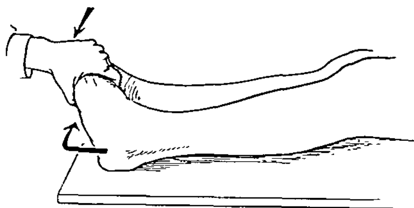


Fig. 27.—Plantar flexion of the foot is performed mainly by the gastrocnemius muscle and it should be palpated during attempts of the patient to flex the foot against resistance.



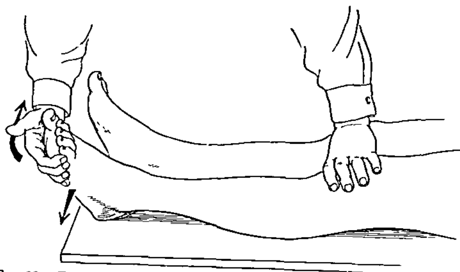


Fig. 28 —To test [illegible]
that position and res[illegible]
actions should be e[illegible] patient's power of eversion of the foot.
Inversion is produced by action of the posterior and anterior tibial muscles and eversion of the foot depends upon the peroneus longus and brevis muscles.

of the *posterior* and *anterior tibial* muscles which are innervated by the posterior tibial and deep peroneal nerves respectively (L4, 5 and S1, 2). Eversion of the foot depends upon the *peronuse longus* and *brevis* which are innervated by the superficial peroneal nerve (L5, S1 and 2). (Fig. 28.)

Dorsiflexion of the toes is carried out by the *extensor hallucis longus* which extends the large toe, the *extensor digitorum longus* which extends all the other toes and the *extensor digitorum brevis* which extends all but the small toe. (Fig. 29.) These muscles are innervated by the deep peroneal nerve. Plantar flexion of the toes, except the large one, is accomplished by contraction of the *flexors digitorum*

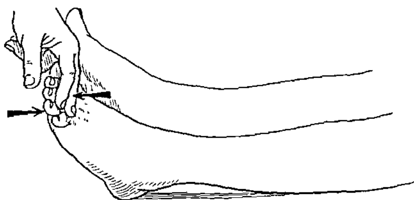


Fig. 29.—Dorsiflexion of the toes is carried out by the extensor hallucis longus which extends the large toes, the extensor digitorum longus which extends all the other toes and the extensor digitorum brevis which extends all but the small toe

longus and *brevis*. The longus acts at the distal interphalangeal joints and the brevis at the metatarsophalangeal and proximal interphalangeal joints. In addition the lumbricals and interossei flex the toes at the metatarsophalangeal and extend them at the interphalangeal joints. The large toe is flexed at the metatarsophalangeal joint by the *flexor hallucis brevis* and at the intertarsal joint by the *flexor hallucis longus*. The patient should flex the toes and attempt to maintain the position against resistance. These muscles are supplied by the posterior tibial nerve (S1 and 2).

Various diseases of the nervous system produce increased rather than reduced motor activities of the body. These hyperkinetic movements all prove to be far in excess of the patient's purpose. *Tremors* are involuntary, rhythmic, oscillatory movements which affect all of the muscles or groups of muscles. They may be present at rest or may be brought out only when an intentional purposeful movement is made. They vary in the speed and range of movement The most satisfactory method of testing for the presence of a tremor

is to have the patient attempt to pour water from one test tube into another with the arms outstretched and without touching the edges of the tubes. Tremor in the face and tongue is best elicited by having the patient, with his eyes closed, vigorously protrude his tongue. *Spasms* may be tonic or clonic. Tonic spasms are involuntary, sustained, muscular contractions and relaxations of muscles or muscle groups. *Convulsions* are continuous or repeated spasms of the clonic or tonic type which are more or less widely distributed. In local or Jacksonian convulsions the movements are confined to a definite portion of the muscular system, such as the face, hand, arm, or lower extremity. General convulsions involve the entire musculature and are usually associated with a disturbance or abolition of consciousness. *Tetany* is characterized by tonic spasms of slow onset and of a peculiar distribution (carpopedal spasm) occurring in attacks and accompanied by pain. Involuntary, quick, incoördinate movements which are conscious but aimless, inimitable, and unphysiological characterize *chorea* and the choreiform diseases. A *tic* is a purposeful, voluntary, imitable movement which may be controlled by volition and modified by restraining gestures and which does not interfere with function. *Athetosis*, a condition which frequently follows a hemiplegia, is characterized by a constant recurring series of slow, vermicular movements of the hands and feet, beginning with hyperextension and always assuming the same pattern.

SENSATION

Disturbances of sensation are prominent objective symptoms in many central nervous system disorders. All forms of sensation may be lost to a varying degree or there may be a dissociation of sensation to the extent that one type of sensory stimuli is recognized and other sensations are lost. Or, all forms of stimuli may be recognized, but the patient is unable to recognize the difference in degrees of those stimuli.

The patient should be examined for a disturbance in his ability to appreciate the stimuli of light touch, pressure touch, superficial and pressure pain, heat and cold, vibration, change in posture of a part of an extremity, and passive movement of a segment of an extremity. One should also determine the patient's ability to recognize the size, shape, and form of objects as well as his stereognostic sensibility.

Light touch sensation is examined for by gently applying a wisp of cotton to the skin. Pressure touch may be tested by the use of the unsharpened end of a lead pencil. Superficial pain may be investigated by pricking with a sharp pin or needle. The succession

of sharp stimuli is interrupted by the application of a dull object, and the patient is asked to differentiate between "sharp" and "dull" each time he is stimulated. A simple algesimeter may be made by placing a large headed pin in the barrel of a glass syringe, allowing the point to extend through the tip and replacing the plunger. The weight of the plunger determines the degree of pressure when the pin is allowed to prick the skin, with the syringe held by the barrel. Pressure pain sensibility may be tested by the use of an instrument with a blunt end to which a definite number of kilograms of pressure may be applied. (Fig. 30.)

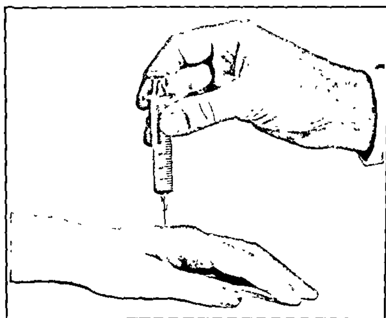


FIG. 30 —Simple method of testing pin-prick sensation

For the purpose of the scientific investigation of temperature sensibility the employment of glass tubes containing hot and cold water is open to objections, but this method suffices for the clinical examination. Cold sensation also be tested using a wisp of cotton dipped in ration sense

designate its direction. The appreciation of size, shape, and form may be tested by the use of various geometric forms. Stereognostic sensibility, or the recognition and naming of familiar objects placed in the hand, may be tested by the use of any common objects, the name of which are familiar to the patient such as coins, a knife, a safety-pin or a key.

Somatic sensations are transmitted from their point of origin over afferent fibers in the peripheral nerves. These fibers are the distal branches of T-shaped axons, whose cells of origin are in the spinal ganglia. The central portions of these axons enter the spinal cord through the posterior spinal root. Each posterior spinal root consists of a large medial and a smaller lateral division. The fibers of the medial division are of a large caliber; they are myelinated, and they run over the tip of the posterior gray horn of the spinal cord into the posterior column of the white matter. The fibers of the lateral division are fine, unmyelinated, and enter the tract of Lissauer which lies along the apex of the posterior gray horn. (Fig. 31.)

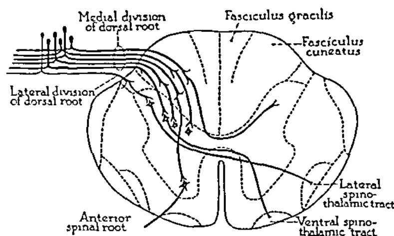


FIG 31.—Diagram which shows entrance of the sensory fibers into the spinal cord and the important tracts in which they ascend.

Separate fibers transmit the afferent impulses of touch, thermal, and pain sensibility within the spinal cord This separation occurs when the posterior spinal root fibers enter the spinal cord. Tactile sensation is carried by the large, myelinated fibers which ascend within the spinal cord into two different pathways. Some of these fibers pass upward in the posterior white columns of the same side while *other fibers cross the median line in the anterior commissure* of white matter and ascend in the anterior column of white matter as the anterior spinothalamic tract. The second station in the tactile pathways is the thalamus and from it neurons of the third order pass to the sensory cortex of the cerebrum. The unmyelinated, fine fibers of the lateral division of the posterior spinal root almost immediately end in the gray matter of the posterior gray horn. Secondary neurons then cross the median line and ascending in the lateral spinothalamic tract to end in the thalamus. Neurons of the third order pass to the postcentral gyrus of the cerebral cortex by way of the thalamic radiation and the posterior limb of the internal capsule. This pathway serves for the transmission of pain and temperature sensations. Sensa-

tions of passive movement, posture, and vibration are transmitted in large myelinated fibers which pass upward in the posterior columns of the white matter of the same side, similar to the pathways for a portion of the tactile impulses.

The thalamus may be compared to a telephone station which handles incoming and outgoing impulses. It is the end station for secondary neurons which arise within the spinal cord, and at the same time it is the point of origin for the fibers which transmit these impulses from the thalamus to the cortex. Impulses which do not terminate within the thalamus are transmitted onward in fibers which pass to the postcentral gyrus through the posterior limb of the internal capsule.

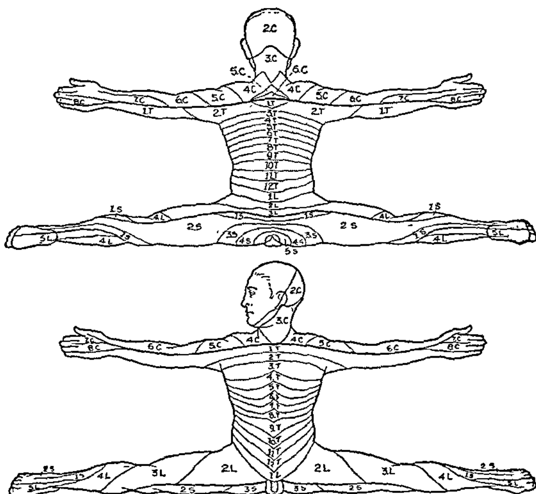


FIG 32 —Diagram to show the segmental distribution of sensation over the body

These various pathways for the transmission of sensation make it obvious that a given lesion within the spinal cord might be characterized by a dissociation of sensation, as contrasted to a lesion of a peripheral nerve which would result in a loss of all forms of somatic sensation. Both are in contrast to the lack of discriminative ability in lesions of the cortical sensory area.

The employment of the various tests previously described make evident intensification and diminution of sensation which are termed respectively, *hyperalgesia*, *hyperesthesia*, *hypalgesia*, and *hypesthesia*. Subjective sensory disturbances, such as numbness, prickling, formication, tingling, and sensations of heat and cold, are known as *paresthesias*. They may exist without any objective loss of sensibility. The subjective presence of pain should involve a careful investigation as to its character, distribution, maximum point, and the accompaniment of tenderness. Lightning pains in tabes, burning and cramp-like pains of neuritis, aching pains of arthritis, and the girdle pains of spinal disease are almost pathognomonic.

It will be remembered that the body is made up of a series of segments represented in the skeleton, muscles, viscera, and nervous system. Thus, upon the skin, may be represented the segmental sensory distribution of the posterior roots of the spinal cord. If one imagines the body in the all-fours position of our ancestors and then makes transverse sections of the body at regular intervals, beginning at the neck and passing to the coccyx, the segmental distribution of the spinal cord will be more clearly understood. In this position it will be readily seen that the corresponding sides of the arms and legs will be sectioned at a higher level than will be the opposite sides. For this reason, the radial sides of the upper extremities are represented by a higher segmental level of the spinal cord than are the ulnar aspects, and in the same manner the medial aspects of the thighs and legs are of a higher segmental level than are the external sides of the lower extremities. The successive segmental levels in the dorsal region are easily understood, and as one passes caudally the segmental representation of the primitive tail is about the anus. It is at once apparent that a loss of sensation corresponding to a segmental area is diagnostic of a spinal cord lesion. (Fig. 32.)

REFLEXES

The spinal reflex mechanism plays the third important rôle in the diagnosis of neurological lesions. The simplest form of the reflex arc consists of a primary sensory and motor neuron and a synapse in the anterior gray matter of the spinal cord. Reduced to its elements the arc is made up of (1) a receptor, or peripheral sensory nerve endings; (2) an afferent conductor; (3) a synaptic center; (4) an efferent conductor; and (5) the effector mechanism or muscle fibers. One or more sensory or motor neurons interposed between the primary neurons make the reflex arc more complex. Neurons which pass through more than one segment of the cord before completing the arc constitute the afferent and efferent limbs of intersegmental reflex arcs. (Fig 33.)

Such more or less complicated arcs are used clinically. Each of these reflexes has its center within a given segment of the spinal cord. Certain reflexes involve the stimulation of muscle tendons, such as the patellar, Achilles, biceps, and triceps, and are termed *deep reflexes*. Others are produced by cutaneous stimulation, such as the abdominal and cremasteric reflexes, and comprise the *superficial reflexes*. Normal activity of a reflex requires the integrity of the center and both limbs of the arc. In addition, there must be a proper association between the reflex center and the cerebrum. Destruction of either limb of the arc abolishes the reflex response.

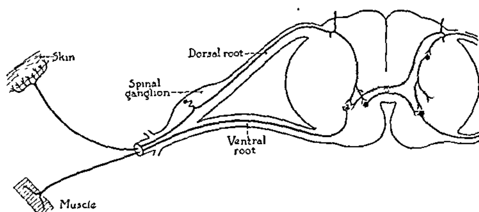


Fig 33.—A diagram of a simple reflex arc.

A lesion which passes through a given spinal cord segment abolishes the reflex having its center at that point. Below the level of the lesion the deep reflexes are increased by removal of inhibition from centers above the lesion, but the deep reflexes having their centers above the level of the pathology are unchanged. The reflex arc for the superficial reflexes probably reaches the cortex, a fact which is offered in explanation of their absence in upper motor neuron lesions. The group of pathological reflexes occur in the presence of lesions of the pyramidal tracts. Extension of the large toe with fanning of the small toes upon plantar stimulation is known as the Babinski phenomenon and is an example of a *pathological reflex*. The Oppenheim, Gordon, and Chaddock phenomena are only modifications in the method of producing such a reflex. The presence of generally exaggerated deep reflexes is without significance in the consideration of an organic lesion unless accompanied by diminished or absent superficial reflexes, pathological reflexes, or unless they definitely give rise to a sustained clonus

The sphincter muscles of the bladder and anus reflexly contract upon the contact of a foreign body. Disturbances of the *sphincteric*

reflex of the bladder may be evidenced by difficulty in starting the urinary stream, retention of urine, or complete incontinence. The *pupillary reflexes* also form important aids in diagnosis. The normal eye shows a decided pupillary contraction if a beam of light is thrown into it, and this is known as the pupillary reflex to light. When focused upon a near object the pupil also contracts, and this is the pupillary reflex to accommodation. If the pupil responds reflexly to accommodation but is quite unresponsive to light, the Argyll Robertson pupil is said to be present.

Below is a table of the various reflexes with their centers:

	Center	
Deep reflexes:		
Jaw-jerk	Cranial n. V	Pons
Triceps	C VI–VII	Spinal cord
Biceps	C V–VI	Spinal cord
Wrist-jerk	C VII–VIII	Spinal cord
Knee-jerk	L II–III	Spinal cord
Achilles' jerk	L V–S I	Spinal cord
Superficial reflexes:		
Cremaster	L I–II	Spinal cord
Plantar	S I	Spinal cord
Upper abdominal	D VIII–X	Spinal cord
Lower abdominal	D X–XII	Spinal cord
Gluteal	L IV, V–S I	Spinal cord
Pathological reflexes:		
Babinski phenomenon		
Oppenheim phenomenon		
Gordon phenomenon		
Chaddock phenomenon		
Sphincteric reflexes:		
Bladder		Spinal cord
Anus		Spinal cord
Pupillary reflexes:		
Reaction to light		
Reaction to accommodation		
Ciliospinal	C VIII	Spinal cord

SPINAL CORD

It is possible now to visualize the location of these few important tracts in a cross-section of the spinal cord. In the lateral columns of the white matter, the crossed pyramidal, or motor tracts descend. Just anterior and more lateral to these fibers are the lateral spinothalamic tracts which carry impulses of pain and temperature upward from the opposite side of the body. In the posterior white columns ascend, uncrossed, the fibers which transmit joint, muscle,

bone, and tactile sensibility. In the anterior gray horns are the large pyramidal cells which give rise to motor axons which reach the somatic muscles. (Fig. 34.)

How far can the inexpert person, untrained in neurology, utilize the information thus far presented for the benefit of his patient? Let it be supposed that the patient has a paralysis of his lower extremities. His limbs are flaccid, his knee-jerks and ankle-jerks are absent, and there is an atrophy of muscles. Immediately an upper motor neuron lesion is ruled out of consideration because such a lesion would be followed by spastic paralysis and increased deep reflexes. An exception should be made for the early stages following

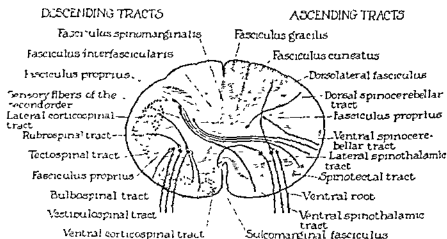


FIG. 34.—Diagram of a cross-section of the spinal cord showing location of the ascending and descending fiber tracts.

a traumatic lesion of the spinal cord in which flaccidity of the muscles is present. Where are lower motor neuron lesions produced? In the anterior gray horn cells of the cord, in the anterior spinal roots, and in the peripheral nerves. If sensory changes are absent, the peripheral nerves may be excluded, leaving the anterior horn cells and the anterior spinal roots. Of these two lesions, the former is the more common by far. One may go further and say that the lesion is due to an anterior poliomyelitis which, if acute, is the common infantile paralysis.

A lesion which affects the pyramidal tracts will produce a paralysis. Inasmuch as this paralysis is of the upper motor neuron type, it will produce increased deep tendon reflexes. Likewise, as the result of diminished cerebral inhibition, muscle tone is increased and spasticity is present in the paralyzed muscles. The separation of the lower motor neuron from the influence of upper levels in the nervous system releases certain protective reflexes which consist of integrated

motor movements. One of these pathological reflexes is Babinski's sign. If, therefore, one sees a patient who has a bilateral spastic paralysis, with increased deep reflexes, with no sensory or trophic disturbances, and with a bilateral Babinski sign, the condition can be due only to bilateral involvement of the pyramidal tracts.

If a patient has a spastic paralysis in the legs with increased deep reflexes and bilateral Babinski phenomena (an upper motor neuron lesion), and in the upper extremities, a flaccid paralysis with a reaction of degeneration, muscle atrophy, and an absence of deep reflexes with no sensory changes (a lower motor neuron lesion), it is obvious that a combined lesion of the lateral white columns and anterior gray horns exists. There is but one disease which commonly produces this group of symptoms and that is amyotrophic lateral sclerosis.

If the posterior white columns, which subserve deep sensibility, were destroyed, an ataxia sensory in character would be produced. The patient would be wholly unable to orient himself concerning the position of his extremities and the extent and direction of their movements. A patient with such a sensory ataxia who has, in addition, an absence of the deep tendon reflexes, with or without pain, and some loss of tactile or pain sensibility has suffered an interruption of the reflex arc in its sensory portion. He has, in other words, a lesion of the posterior spinal roots and the posterior white columns. Such a condition can be the result only of a tabes dorsalis, or locomotor ataxia. The lesion of the posterior spinal root produces an absence of deep reflexes, spontaneous pain, and a loss of sensation. Inasmuch as deep sensibility is of more recent acquisition than pain, the long fibers in the posterior columns first undergo secondary degeneration. This results in the loss of joint and muscle sense and ataxia occurs.

If a patient presents himself with a loss of joint and muscle sense, a sensory ataxia, and a spastic weakness with increased deep tendon reflexes, it is obvious that he has an involvement of more than one column of the spinal cord. He has, in fact, a combined lesion of the posterior and lateral white columns which can be due to a very few diseases. The chief of these is combined degeneration of the spinal cord which is generally caused by pernicious anemia. If in addition to the neurological phenomena, there is a positive color index and an achlorhydria, the diagnosis may become quite positive.

Let us suppose that a patient has a paresis of his lower extremities with increased deep tendon reflexes, bilateral Babinski phenomena, and spasticity; in the upper extremities he has a flaccid paralysis with muscle atrophy, a reaction of degeneration, and absence of the deep tendon reflexes. It would be immediately obvious that *'

lesion affects the anterior gray matter at the level of the upper extremities and presses upon the lateral columns. If it is found upon examination that in the levels of the muscular supply of the upper extremities there is a loss of sensation to pin prick and temperature stimuli but a preservation of tactile sense, the diagnosis is apparent. It will be remembered that the fibers which transmit pain and temperature sense enter the spinal cord and cross in the gray matter to the opposite spinothalamic tract. This dissociation of sensation can be produced only by a lesion about the central gray matter which interrupts the pain and temperature fibers as they cross; whereas, the ascending fibers for tactile sensibility remain unaffected in the posterior white columns. Such a lesion may be due to but two diseases—intramedullary tumor or syringomyelia.

Any disease which produces a transverse lesion of the spinal cord is followed by interruption of the deep reflex arc originating at that segmental level of the spinal cord. At that level there would be a flaccid paralysis with the reaction of degeneration and muscular atrophy. Often this level is necessarily very narrow, and these signs may be difficult to elicit. However, having also interrupted the pyramidal tracts there would be a spastic paralysis with increased deep reflexes, absent superficial reflexes, and pathological reflexes below the level of the lesion. Since the ascending sensory pathways would also be interrupted at the level of the lesion, there would be a loss of sensation below that level. The bladder musculature, which corresponds in a measure to the somatic musculature, would be inhibited by a sphincteric hypertension and a retention of urine would result. Such a transverse lesion may be produced by myelitis, tumor, fracture-dislocation of the spinal column, thrombosis, hemorrhage, abscess, or by diseases of the spine. The onset of a transverse lesion of the cord many times confirms or excludes certain etiological facts. For example, the vascular lesions are sudden in onset while tumors and compressions due to an abscess, or Pott's disease, are usually characterized by slowly developing symptoms

By referring to anatomical facts, considerable assistance may be obtained in the differentiation of transverse lesions of the spinal cord. The fibers for pain and temperature sense, as they enter the spinal cord and cross, ascend in a lamellar manner. Those fibers from the lowermost segments are gradually pushed outward by the increasing number of fibers from the upper segments. Thus, in an incomplete intramedullary lesion in the cervical region, the outermost part of the spinothalamic tract is intact, and as this represents the lowermost segments it will be found that the saddle area supplied by the sacral segments remains unaffected. On the other hand, in

a lesion which produces compression from without, the lowermost segments are first affected, so that there is an analgesia up to, but not including, the last three or four segments below the level of the lesion.

There is a corresponding assistance afforded in the diagnosis of diseases of the brain stem, cerebrum, cerebellum, and cranial nerves by the possession of even a small amount of working knowledge concerning the anatomy and physiology of these structures. There are twelve pairs of cranial nerves, and the functions of the majority of them can be tested for without much difficulty.

CRANIAL NERVES

Olfactory.—Each naris should be tested separately for the presence of the sense of smell. Pungent odors should be avoided in making such tests because they stimulate general sensation. A complete absence of the sense of smell is termed *anosmia.*

In the olfactory portion of the mucous membrane of the nose are bipolar sensory cells. The central prolongations of these cells gathered into small bundles constitute the olfactory nerves which almost immediately enter the olfactory bulb Within the glomeruli of the olfactory bulb the impulses are transferred to the dendrites of mitral cells. From these cells impulses are carried in the lateral olfactory striæ to the pyriform area of the cerebrum. Other fibers carry the olfactory impulses to the hippocampus and fascia dentata.

Optic.—In addition to the pupillary reflexes, a careful examination of the eyes will reveal many important facts. The fields of vision and ophthalmoscopic examination of the optic fundi often yield valuable diagnostic information

The visual field, or the area over which objects are visible with fixation of the eye, may be tested by the use of color or form test objects The use of various sized and shaped objects is a much more accurate method of determining the visual fields since they are perceived before colors are apparent For an accurate determination of the visual field an arc or screen perimeter is necessary, but gross defects in the field of vision can be found by a simple examination. The examiner and patient should sit facing each other at a distance of about 2 feet. When testing the left eye, the patient should hold his right hand over his right eye, and the examiner should cover his own left eye The patient should then fix his left eye upon the examiner's right pupil A small white piece of paper upon the end of a pencil or wooden applicator may serve as a test object This is moved on a straight line midway between the patient and examiner, and the field of vision is outlined by moving the test

object from the periphery in a vertical and horizontal plane. If the examiner knows the limits of his own field of vision, any abnormality in the patient's field may be easily ascertained.

It will be remembered that because of their embryological development the retinæ resemble the brain structure. Visual impulses are received through the rods and cones, the highly differentiated receptor endings of the visual cells of the retina, and are transmitted to the ganglion cells. Axons of these cells transmit the impulses by way of the optic nerve to the superior colliculus, lateral geniculate body, and the pulvinar of the thalamus. The optic nerves unite after their entrance into the cranial cavity and form the optic chiasm where a decussation occurs. The fibers from the temporal halves of the retinæ pass back uncrossed, while the nasal fibers cross to the opposite side and unite with the temporal fibers to form the optic tract. The optic tracts partially encircle the cerebral peduncles to reach the lateral geniculate body, pulvinar of the thalamus, and the superior colliculus. Fibers concerned only with reflex visual impulses are contained within the optic tracts as far as the superior colliculus.

From the lateral geniculate body and the pulvinar of the thalamus fibers arise which pass back through the posterior limb of the internal capsule into the occipital lobe. These fibers form the optic radiation. The visual cortical center in the occipital lobe lies mainly on the medial aspect and is divided by the calcarine fissure into an upper and lower portion. The lower quadrants [illegible] bove the fissure [illegible] in the lingual gyrus below the calcarine fissure

Objects to the left of the visual axis are reproduced on the nasal side of the left retina and the temporal side of the right, and because of the decussation these impulses reach the right visual cortical center. In the same way objects in the right visual field reach the left visual cortex. Destruction of one optic nerve anterior to the decussation produces total blindness in that eye, while a lesion in the optic tract or in the optic radiations of the corresponding visual cortex produces a blindness in both eyes for the opposite lateral half of the visual field. Such a defect in the visual field is known as *hemianopsia*. Blindness in both temporal fields of vision or both nasal fields is known as *bitemporal* or *binasal hemianopsia*, as the case may be The former is commonly produced by pressure upon the optic chiasm by a pituitary tumor. A lesion of the optic radiations may give rise to a *quadrantic anopsia*, in which only one-quarter of the field, either upper, lower, nasal, or temporal is lost. Such defects may be determined only by the use of the quantitative perimeter. A *scotoma* may be defined as a spot of varying size, shape, and intensity within the visual field, in which there is no vision. The *blind spot*, the point of entrance of the optic nerve, is considered as a physiological scotoma Other scotomas are classified as to shape, being annular when they surround the macula; central when

they are directly in the macula; peripheral when they are outside the macular area; scintillating when they are evanescent; and paracentral when they are partially central but do not entirely obscure central vision. Color scotomas are patches of color blindness in the visual field and are usually central. Scotomas may be negative when they are not ordinarily perceived but are detected only by examination; positive when perceived subjectively; relative when there is partial impairment; and complete or absolute when no vision is obtainable. (Fig. 35.)

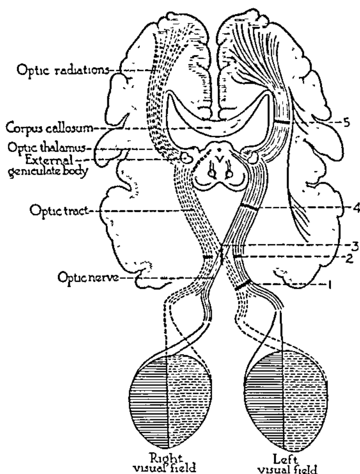


FIG. 35.—Diagram which shows the location of the lesion of various types of blindness: (1) [illegible] (2) [illegible] (3) [illegible] anopsia; (4 [illegible] (5) right ho [illegible]

It will be remembered that the optic nerve fibers cross in the optic chiasm and proceed backward to the optic radiations in the occipital lobe so that a lesion behind the commissure affecting one optic nerve produces a blindness of the corresponding halves of both retinæ. On the other hand, a lesion in the central part of the optic

commissure produces a blindness in the temporal halves of both visual fields.

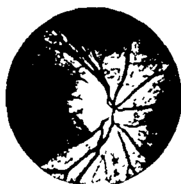
The ultimate diagnosis of many diseases of the nervous system often rests upon an accurate interpretation of an *ophthalmoscopic* examination.

When the *normal fundus* is examined by the ophthalmoscope, one sees a bright red background produced by the chorioid and the circulating blood in the chorioidal vessels. The retina is transparent normally so that no part of it except the blood-vessels is seen. The *papilla* or optic nerve head is circular or oval in shape and of a light grayish or yellowish-red color contrasted with the red of the remainder of the fundus. It lies in the same plane as the retina and does not form a projection. Often the papilla contains a depression in its outer half known as the *physiological excavation*. The blood-vessels come out of the papilla upon the inner border of the excavation, divide, and pass over the edge into the retina where they become arborescent. The arteries and veins may be distinguished readily. The arteries are bright red, narrow, and straight. The veins are darker, larger, and tortuous.

Hyperemia of the retina may be produced by inflammation or by compression of the veins. Extravasations of blood in dark red patches may be seen upon the retina. *Arteriosclerosis* and high vascular tension produce a picture characterized by corkscrew arterial twigs, flattened veins, a dull red congestion or edema of the optic disc, and later white streaks along the arteries and veins. *Albuminuric retinitis* produces haziness of the retina, indistinctness of the edges of the papilla, distention of the retinal arteries, hemorrhages, and yellowish-white exudative patches. The latter are quite characteristic and are found in an area around the papilla and in the macula lutea. They commonly have a stellate arrangement. In some instances retinal hemorrhages and a neuritis may be the only findings.

The pathology present may involve most prominently the *papilla* and produce a *neuritis* or *papilledema*. The latter condition is caused by the transmission of increased intracranial tension to the optic nerve sheath and the resultant incarceration of the optic nerve The mechanism of the production of papilledema (choked disc) is still debatable, but it seems probable that compression of the central retinal vein as it crosses the subdural space, due to the elevation of pressure in that space, is responsible for engorgement of the retinal veins, while compression of the lymph spaces in the nerve itself causes swelling of the nerve head. Intracranial space-occupying lesions are the most common causes of papilledema. The papilla becomes white, gray, or reddish in color, and is often mottled with

PLATE I



Normal



Macular star



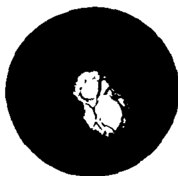
Hypertensive neuroretinitis



Hypertensive neuroretinitis



Optic atrophy (Tabes)



Optic atrophy following papilledema

(Courtesy of Department of Opthalmology Northwestern University Medical School and Dr Robert Von der Heydt)

PLATE I



Optic neuritis



Optic neuritis



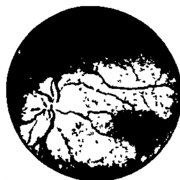
Papilledema



Papilledema



Papilledema



Subsiding papilledema with evidence of atrophy

(Courtesy of Department of Ophthalmology, Northwestern University Medical School and Dr. Robert Von der Heydt)



white spots or extravasations of blood. The outline of the papilla becomes indistinguishable. The arteries of the retina are smaller, and the veins become distended and tortuous due to the compression produced by the swollen optic nerve. The degree of projection of the papilla is calculated from the difference in refraction between it and the retina. In a choked disc the papillary swelling is so great that the vessels appear kinked or interrupted, particularly at the outer border of the papilla. (Plate I.)

Optic *neuritis* means inflammation of the intra-ocular portion of the optic nerve. The nerve head appears hyperemic, its borders are blurred, and it may become elevated. Hemorrhages may occur on the surface of the nerve, and in the nerve fiber layer of the retina about the disc.

Atrophy of the optic nerve may be primary or secondary. The picture of a primary atrophy is that of a pale papilla, white or bluish-white in color, with sharply defined edges. The retinal vessels are unaltered. *Primary optic atrophy* occurs in tabes, multiple sclerosis, and in tumors which exert direct pressure upon the optic chiasm or nerves. *Secondary atrophy* follows neuritis or retinitis. Early, the papilla is grayish-white in color with hazy margins. The retinal veins are distended and tortuous. Later the papilla becomes bluish-white in color, sharply defined, but smaller than normal. *Neuroretinitis* consists of an inflammation of the retina plus involvement of the optic nerve.

Oculomotor, Trochlear, and Abducens.—The movements of the eyeball are accomplished by the extrinsic ocular muscles. With the exception of the superior oblique and external rectus muscles, which are innervated by the trochlear and abducens nerves respectively, all of the extra-ocular muscles are dependent for their nerve supply upon the third cranial nerve. These muscles may become involved singly or in groups, giving rise to an inability to direct both eyes in a given direction, and consequently to disturbances of vision.

Often the patient complains of blurred vision or actually states that he sees two objects when looking in a certain direction. This may be checked by the examiner more carefully by determining whether the double vision is present when the patient looks to the left, right, upward, or downward, and in addition this gives some clue to the muscle involved. Weakness of the extra-ocular muscles should be examined by having the patient follow the examiner's finger tip without moving his head. The finger should be moved to the right, left, downward, and upward. Often one cannot detect a strabismus unless the weakness is very marked, but it can be brought out definitely by having the patient indicate when he sees the examiner's

finger doubly. In a general way it may be said that strabismus occurs in a direction away from the traction of the paralyzed muscles while diplopia occurs in the direction of traction of the paralyzed muscle.

The nuclei of the oculomotor and trochlear nerves are in the midbrain while the nucleus for the abducens nerve lies in the pons The course of the latter nerve from its point of origin to its exit from the skull is therefore a long one, and for that reason it is more subject to trauma. In addition to innervating extra-ocular muscles, the oculomotor nerve supplies fibers to the levator palpebrarum muscle.



FIG 36.—Photograph of patient with ptosis and ophthalmoplegia

Nystagmus is one of the most important localizing symptoms to be found in a neurological examination. It is present in a variety of organic diseases of the nervous system which affect the midbrain, pons, and particularly the cerebellum and vestibular nerve pathways Nystagmus may be present when the eyes assume their spontaneous position, or as is more often the case ' eye are deviated from

Drooping of the upper eyelid, or ptosis, occurs as the result of a paralysis of the levator palpebræ superioris muscle which is innervated by the oculomotor nerve. When ptosis is present, usually all of the other muscles supplied by the third nerve including the constrictor of the iris and the ciliary muscle are also paralyzed. The patient is unable to move the eyeball upward, directly downward, or directly inward, and the pupil is dilated and will not contract to light or accommodation. An aneurysm of the internal carotid artery, syphilis, trauma, or an intracranial tumor about the sella turcica may cause an oculomotor nerve paralysis. Not uncommonly the fourth and sixth nerves are also paralyzed and a complete ophthalmoplegia exists. (Fig. 36.)

Trigeminal.—This is the large cranial nerve which supplies the face with sensation and is divided into ophthalmic, maxillary, and mandibular divisions. The most common disease which affects the trigeminal nerve is a severe, intractable neuralgia, variously known as tic douloureux, trifacial neuralgia, or more accurately, as trigeminal neuralgia. Various intracranial tumors may involve this nerve by pressure or its ganglion may be the site of a neoplastic growth.

The fibers of the three divisions of this nerve originate from cells in the large Gasserian, or semilunar ganglion, which is located in the middle fossa of the skull and which is covered by a dural envelope. Connecting the ganglion to the pons is the large sensory root. Superior and medial to the sensory root is the more compact and smaller motor root. The latter passes beneath the ganglion and emerges from the skull with the mandibular division.

The sensory portion of this nerve carries tactile, painful, and thermal impulses from a large part of the skin and mucous membranes of the head. Cells in the semilunar ganglion give rise to central fibers which enter the pons and divide into ascending and descending branches. The former terminate in the main sensory nucleus, while the latter end in the spinal nucleus. There is reason to believe that pain and thermal sensations are carried in the spinal tract of this nerve which is a direct continuation of the substantia gelatinosa Rolandi of the spinal cord. Secondary fibers arising in the spinal and main sensory nuclei cross the median line, ascend, and terminate in the lateral nucleus of the thalamus From here a tertiary group of fibers run to the posterior central cortical gyrus through the posterior limb of the internal capsule. Dissociation of sensation may occur in the area supplied by the trigeminal nerve as is true in the spinal cord

The motor root passes beneath the semilunar ganglion and becomes incorporated within the mandibular division to supply the masseter, pterygoid, temporal, and other muscles.

A destructive lesion of the trigeminal nerve is followed by a loss of sensation to pin prick, light touch, and temperature stimuli on the corresponding half of the face. It must be remembered that the superficial cervical nerves supply a rather large area of overlap of sensation to the face in the area of distribution of the mandibular

division so that it is well to refresh one's memory of the isolated supply of the trigeminal nerve for sensation. In addition to this loss of sensation, the cornea is insensitive as are the conjunctiva and the mucous membranes of the corresponding side of the nose, mouth, and anterior two-thirds of the tongue. Although the fifth nerve supplies other portions of the mucous membranes the overlap from other cranial nerves is so great that a loss of sensation cannot be made out easily.



FIG. 37 —(*a*) Usual limits of sensory loss in complete division of the sensory root of the trigeminal nerve, (*b*) deviation of the jaw toward the paralyzed side in a lesion of the motor root of the trigeminal nerve.

The methods for testing sensation elsewhere over the body suffice in this instance and have already been described. Often, however, the corneal reflex may be absent upon one side and is the earliest indication of a beginning lesion of the fifth nerve

A lesion of the fifth nerve is also followed by paralysis and atrophy of the masseter and the pterygoid muscles. Because of the paralysis of the pterygoids, when the jaw is opened it deviates toward the side of the paralysis. (Fig 37.)

Facial.—The seventh cranial nerve innervates the facial muscles of expression and its destruction by trauma or by disease is followed by a deforming paralysis of the facial muscles. This cranial nerve is more frequently involved in diseases of the nervous system than any other

When a peripheral paralysis of the facial nerve exists, the face has a one-sided appearance, and the paralyzed side is flat and expressionless As the patient speaks, he appears to talk out of one side of his mouth, and when he blinks his eyes, the lid on the affected side fails to close or lags behind the normal side. If the patient is asked to show his teeth, to smile, or to whistle, there is a distinct asymmetry, and the lips are drawn further to the normal side. If the peripheral lesion of the facial nerve is of long standing, when the

patient laughs spontaneously, this marked asymmetry may all disappear; or if the patient's face is in repose, the asymmetry may be less marked although the difference in the depth of the nasolabial folds is easily made out. The upper portion of the facial muscles may be tested by asking the patient to frown or to shut his eyes tightly. Weakness of the orbicularis oculi muscle may be determined by having the patient attempt to keep his eyes closed against the examiner's efforts to open them. Weakness in this muscle can be determined in a patient unable to coöperate by raising the upper eyelid and then releasing it. On the affected side the lid will cover the eyeball slowly and incompletely; whereas, on the normal side the lid covers the eyeball quickly and with an appreciable measure of muscle tone. (Fig. 38.)



FIG. 38.—The facial muscles may be tested by having the patient show his teeth. The platysma muscle contracts when this is performed vigorously.

There is a loss of taste on the anterior two-thirds of the tongue in the presence of a lesion of the facial nerve central to the chorda tympani branch and peripheral to the geniculate ganglion. This function of the nerve may be tested by placing salty, sweet, or bitter substances upon the protruded tongue and asking the patient to indicate the sensation on a chart or paper without drawing his tongue into his mouth. Perhaps an easier and more accurate method of testing is to touch the tongue with a copper wire electrode connected with a weak galvanic current. Normally this produces a characteristically bitter taste.

The giant pyramidal cells of the lowermost part of the motor cortex give rise to fibers which run through the genu of the internal capsule and the cerebral peduncles to end about the nucleus of the seventh nerve in the pons. The majority of these fibers end about the nucleus of the opposite side, but some

end in the nucleus of the same side. From the motor nucleus of the facial nerve, which extends from the lower border of the pons to the level of the facial colliculus, fibers arise which partly encircle the nucleus of the abducens nerve and then emerge between the spinal tract of the trigeminal nerve and the facial nucleus. After leaving the pons the facial nerve with its sensory portion, or pars intermedia, enters the internal auditory meatus with the *acoustic nerve and passes through the stylomastoid foramen to supply* the muscles of the face. The geniculate ganglion is formed on the facial nerve in the canal where the nerve turns backward. From this ganglion the greater superficial petrosal nerve arises to pass forward to the sphenopalatine ganglion. Behind the tympanum the chorda tympani branch is given off, and after passing beneath the external pterygoid muscle it becomes incorporated with the lingual branch of the mandibular division of the trigeminal nerve to be distributed to the anterior two-thirds of the tongue. Taste impulses from this portion of the tongue are carried cranialward through the chorda tympani *and nervus intermedius to reach the tractus solitarius in the brain stem.*

The difference between a lesion of the facial nerve above and below the nucleus should be kept clearly in mind because of its importance in making a diagnosis. In an infranuclear or peripheral lesion, such as occurs in Bell's palsy, or injury to the nerve in the bony facial canal, all of the muscles of expression on the corresponding side of the face are involved. In other words, the patient cannot wrinkle his forehead, close his upper eyelid or pucker his lips. In a supra*nuclear lesion, only the muscles of the lower part of the face, that is* from the lower eyelid downward, are involved. The few corticobulbar fibers which do not cross to the opposite side supply the muscles of the upper part of the face in addition to the fibers from the opposite side of the cerebrum. Therefore, in a supranuclear lesion the uncrossed fibers to the frontalis and orbicularis oculi muscles remain untouched. (Fig. 39.)



Fig. 39 Typical appearance of a patient with a right infranuclear facial paralysis.

Supranuclear lesions of the facial nerve occur in intracranial tumors, abscesses, vascular lesions, or other destructive lesions of the cerebrum. Infranuclear lesions occur as the result of direct injury to the nerve during the course of mastoid operations, following skull fractures, pressure by tumors in the cerebello-pontile angle, following exposure to cold, or as the result of infection.

Acoustic. The eighth cranial nerve consists of two portions. The auditory or cochlear is the nerve of hearing, and the vestibular portion is the nerve of equilibrium.

The sense of hearing may be tested quite satisfactorily in a very simple manner by having the patient indicate the distance at which the tick of a watch can be heard. The two sides should be compared with each other and with that of the examiner. If a defect is present, then one should determine whether or not the deafness is due to a lesion of the conduction or the nerve apparatus. A tuning fork is set vibrating, and the end of the fork is placed firmly against the tip of the mastoid process. The patient is asked to indicate when he ceases to hear the musical note. At this point the fork, which should still be vibrating, is held close to the external auditory meatus and again the patient indicates when the note is no longer heard. In the normal individual the note is heard by air conduction about twice as long as it is by bone conduction (Rinne's test). In disease of the middle ear, bone conduction is greater than air conduction; that is, the normal relationship is reversed. In disease of the auditory nerve the normal relationship is maintained, but there may be a pronounced decrease in the duration of time the note is heard. If the end of a vibrating tuning fork is placed on the vertex of the skull, in the presence of a diseased auditory nerve, the sound is heard better on the normal side. In other words, if the note is lateralized in this test (Weber) an impairment of hearing exists. A more accurate method of determining acuity of hearing is to test the patient upon an audiometer and determine his ability to hear tuning forks whose vibrations vary widely in range.

Often patients with neurological lesions complain of tinnitus or noises in the ear, which indicate irritation of some portion of the hearing apparatus. This may be due to an intracranial tumor, aneurysm, or to such simple conditions as wax on the tympanic membrane, or a middle-ear disease. For example, in a tumor of the eighth nerve (acoustic neurinoma) tinnitus is one of the first symptoms, and in labyrinthitis it is one of the most annoying symptoms, while in aneurysms it may be the only subjective symptom. Patients describe their tinnitus in a particularly individualistic manner To some it is the noise of hissing, escaping steam; to others, "a hammer pounding on an anvil"; "ticking of a watch"; or, the "noise of running water."

The auditory nerve conveys impulses produced by sound waves which reach the cochlea Fibers which arise in the spiral ganglion of the cochlea terminate in a dorsal and ventral cochlear nucleus located in the restiform body near the point where it turns into the cerebellum. Fibers of the secondary pathway arise from these nuclei, pass medially as the trapezoid body, cross the median line and form the lateral lemniscus, in which they pass cephalad. Some of the fibers then end in the superior olivary nucleus where they make connections with other fibers belonging to a reflex pathway which connects the cochlear nerve with the nuclei of the motor nerves of the head

end in the nucleus of the same side. From the motor nucleus of the facial nerve, which extends from the lower border of the pons to the level of the facial colliculus, fibers arise which partly encircle the nucleus of the abducens nerve and then emerge between the spinal tract of the trigeminal nerve and the facial nucleus. After leaving the pons the facial nerve with its sensory portion, or pars intermedia, enters the internal auditory meatus with the acoustic nerve and passes through the stylomastoid foramen to supply the muscles of the face. The geniculate ganglion is formed on the facial nerve in the canal where the nerve turns backward. From this ganglion the greater superficial petrosal nerve arises to pass forward to the sphenopalatine ganglion Behind the tympanum the chorda tympani branch is given off, and after passing beneath the external pterygoid muscle it becomes incorporated with the lingual branch of the mandibular division of the trigeminal nerve to be distributed to the anterior two-thirds of the tongue. Taste impulses from this portion of the tongue are carried cranialward through the chorda tympani and nervus intermedius to reach the tractus solitarius in the brain stem.

The difference between a lesion of the facial nerve above and below the nucleus should be kept clearly in mind because of its importance in making a diagnosis. In an infranuclear or peripheral lesion, such as occurs in Bell's palsy, or injury to the nerve in the bony facial canal, all of the muscles of expression on the corresponding side of the face are involved In other words, the patient cannot wrinkle his forehead, close his upper eyelid or pucker his lips. In a supranuclear lesion, only the muscles of the lower part of the face, that is from the lower eyelid downward, are involved. The few corticobulbar fibers which do not cross to the opposite side supply the muscles of the upper part of the face in addition to the fibers from the opposite side of the cerebrum. Therefore, in a supranuclear lesion the uncrossed fibers to the frontalis and orbicularis oculi muscles remain untouched. (Fig 39.)



Fig 39 Typical appearance of a patient with a right infranuclear facial paralysis

Supranuclear lesions of the facial nerve occur in intracranial tumors, abscesses, vascular lesions, or other destructive lesions of the cerebrum. Infranuclear lesions occur as the result of direct injury to the nerve during the course of mastoid operations, following skull fractures, pressure by tumors in the cerebello-pontile angle, following exposure to cold, or as the result of infection.

Acoustic. The eighth cranial nerve consists of two portions The auditory or cochlear is the nerve of hearing, and the vestibular portion is the nerve of equilibrium.

The sense of hearing may be tested quite satisfactorily in a very simple manner by having the patient indicate the distance at which the tick of a watch can be heard. The two sides should be compared with each other and with that of the examiner. If a defect is present, then one should determine whether or not the deafness is due to a lesion of the conduction or the nerve apparatus. A tuning fork is set vibrating, and the end of the fork is placed firmly against the tip of the mastoid process. The patient is asked to indicate when he ceases to hear the musical note. At this point the fork, which should still be vibrating, is held close to the external auditory meatus and again the patient indicates when the note is no longer heard. In the *normal individual the note is heard by air conduction about* twice as long as it is by bone conduction (Rinne's test). In disease of the middle ear, bone conduction is greater than air conduction; that is, the normal relationship is reversed. In disease of the auditory nerve the normal relationship is maintained, but there may be a pronounced decrease in the duration of time the note is heard. If the end of a vibrating tuning fork is placed on the vertex of the skull, in the presence of a diseased auditory nerve, the sound is heard better on the normal side. In other words, if the note is lateralized in this test (Weber) an impairment of hearing exists. A more accurate method of determining acuity of hearing is to test the patient upon an audiometer and determine his ability to hear tuning forks whose vibrations vary widely in range.

Often patients with neurological lesions complain of tinnitus or noises in the ear, which indicate irritation of some portion of the hearing apparatus. This may be due to an intracranial tumor, aneurysm, or to such simple conditions as wax on the tympanic membrane, or a middle-ear disease. For example, in a tumor of the eighth nerve (acoustic neurinoma) tinnitus is one of the first symptoms, and in labyrinthitis it is one of the most annoying symptoms, while in aneurysms it may be the only subjective symptom. Patients describe their tinnitus in a particularly individualistic manner. To some it is the noise of hissing, escaping steam; to others, "a hammer pounding on an anvil"; "ticking of a watch"; or, the "noise of running water."

The auditory nerve conveys impulses produced by sound waves which reach the cochlea. Fibers which arise in the spiral ganglion of the cochlea terminate in a dorsal and ventral cochlear nucleus located in the restiform body near the point where it turns into the cerebellum. Fibers of the secondary pathway arise from these nuclei, pass medially as the trapezoid body, cross the median line and form the lateral lemniscus, in which they pass cephalad. Some of the fibers then end in the superior olivary nucleus where they make connections with other fibers belonging to a reflex pathway which connects the cochlear *nerve with the nuclei of the motor nerves of the head*

end in the nucleus of the same side. From the motor nucleus of the facial nerve, which extends from the lower border of the pons to the level of the facial colliculus, fibers arise which partly encircle the nucleus of the abducens nerve and then emerge between the spinal tract of the trigeminal nerve and the facial nucleus After leaving the pons the facial nerve with its sensory portion, or pars intermedia, enters the internal auditory meatus with the acoustic nerve and passes through the stylomastoid foramen to supply the muscles of the face. The geniculate ganglion is formed on the facial nerve in the canal where the nerve turns backward. From this ganglion the greater superficial petrosal nerve arises to pass forward to the sphenopalatine ganglion. Behind the tympanum the chorda tympani branch is given off, and after passing beneath the external pterygoid muscle it becomes incorporated with the lingual branch of the mandibular division of the trigeminal nerve to be distributed to the anterior two-thirds of the tongue. Taste impulses from this portion of the tongue are carried cranialward through the chorda tympani and nervus intermedius to reach the tractus solitarius in the brain stem.

The difference between a lesion of the facial nerve above and below the nucleus should be kept clearly in mind because of its importance in making a diagnosis. In an infranuclear or peripheral lesion, such as occurs in Bell's palsy, or injury to the nerve in the bony facial canal, all of the muscles of expression on the corresponding side of the face are involved. In other words, the patient cannot wrinkle his forehead, close his upper eyelid or pucker his lips. In a supranuclear lesion, only the muscles of the lower part of the face, that is from the lower eyelid downward, are involved. The few corticobulbar fibers which do not cross to the opposite side supply the muscles of the upper part of the face in addition to the fibers from the opposite side of the cerebrum. Therefore, in a supranuclear lesion the uncrossed fibers to the frontalis and orbicularis oculi muscles remain untouched. (Fig. 39.)



Fig. 39 Typical appearance of a patient with a right infranuclear facial paralysis

Supranuclear lesions of the facial nerve occur in intracranial tumors, abscesses, vascular lesions, or other destructive lesions of the cerebrum. Infranuclear lesions occur as the result of direct injury to the nerve during the course of mastoid operations, following skull fractures, pressure by tumors in the cerebello-pontile angle, following exposure to cold, or as the result of infection.

Acoustic. The eighth cranial nerve consists of two portions. The auditory or cochlear is the nerve of hearing, and the vestibular portion is the nerve of equilibrium.

The sense of hearing may be tested [illegible] simple manner by having the patient [illegible] the tick of a watch can be heard. The [illegible] with each other and with that of the exam[illegible] then one should determine whether [illegible] lesion of the conduction or the nerve appara[illegible] set vibrating, and the end of the fork is [illegible] tip of the mastoid process. The patient is asked [illegible] ceases to hear the musical note. At this point [illegible] still be vibrating, is held close to the external a[illegible] again the patient indicates when the note is [illegible] the normal individual the note is heard by a[illegible] twice as long as it is by bone conduction (Rinne's test [illegible] of the middle ear, bone conduction is greater than [illegible] that is, the normal relationship is reversed. In disease [illegible] nerve the normal relationship is maintained, but there [illegible] pronounced decrease in the duration of time the note [illegible] the end of a vibrating tuning fork is placed on the vertex [illegible] skull, in the presence of a diseased auditory nerve, the [illegible] better on the normal side. In other words, if the note is lateral[illegible] in this test (Weber) an impairment of hearing exists. A more [illegible] method of determining acuity of hearing is to test the patient [illegible] an audiometer and determine his ability to hear tuning forks [illegible] vibrations vary widely in range.

Often patients with neurological lesions complain of tinnitus [illegible] noises in the ear, which indicate irritation of some portion of the hearing apparatus. This may be due to an intracranial tum[illegible], aneurysm, or to such simple conditions as wax on the tympan[illegible] membrane, or a middle-ear disease. For example, in a tumor of the eighth nerve (acoustic neurinoma) tinnitus is one of the first symp toms, and in labyrinthitis it is one of the most annoying symptoms, while in aneurysms it may be the only subjective symptom. Patients describe their tinnitus in a particularly individualistic manner. To some it is the noise of hissing, escaping steam; to others, "a hammer pounding on an anvil"; "ticking of a watch"; or, the "noise of running water."

The auditory nerve conveys impulses produced by sound waves which reach the cochlea Fibers which arise in the spiral ganglion of the cochlea terminate in a dorsal and ventral cochlear nucleus located in the restiform body near the point where it turns into the cerebellum. Fibers of the second-ary pathway arise from these nuclei, pass medially as the trapezoid body, cross the median line and form the lateral lemniscus, in which they pass cephalad. Some of the fibers then end in the superior olivary nucleus where they make connections with other fibers belonging to a reflex pathway wh[illegible] connects the cochlear nerve with the nuclei of the motor nerves of the [illegible]

and neck. The main body of fibers in the lateral lemniscus finally reach the inferior colliculus and the medial geniculate body. The latter functions as a way station to the cerebral auditory center in the temporal lobe. Just as the superior colliculus constitutes the reflex center for visual impulses, so does the inferior colliculus represent the auditory reflex center.

Disease of the vestibular division of the acoustic nerve produces symptoms which may be observed more definitely and estimated more accurately than those which arise from involvement of the cochlear division. These symptoms are vertigo, nystagmus, and ataxia, all of which are also found in lesions of the cerebellum. Nystagmus, or oscillation of the eyes, due to disease of the vestibular mechanism, consists of a quick and slow component. The slow component is the result of a steady stream of nerve impulses from the vestibular system. The quick component comes from the cerebrum and periodically interrupts the vestibular pull; thus causing nystagmus, which may be described as horizontal, vertical, diagonal, rotary, or mixed horizontal and rotary. The most characteristic sensation of vertigo is the apparent rotation of the patient or of external objects about the patient, and this must be differentiated from the subjective symptom "dizziness" of which many patients complain. The hydrostatic conditions present in the three semicircular canals lead to the origination of nervous impulses in the vestibular nerve. If there be a contradiction between the stimuli received from the vestibular apparatus and the actual position of the body in space, the impulses received from the muscles, joints, and eyes will give rise to rotary imaginary movements. In walking, with the eyes closed, patients with vestibular disease deviate toward the side of the lesion and walk as if they were intoxicated.

Impulses from the semicircular canals give messages about rotational movements of the body in space. Impulses from the maculæ of each sacculus and utriculus give messages about linear movements in space and tell the position of the head They also control certain reflexes to the eyes and limbs and influence the tone of muscle. These impulses are carried by fibers which arise from the vestibular ganglion in the internal auditory meatus and pass cephalad through the vestibular nerve to five vestibular nuclei, one of which is the cerebellum, and may be considered as a highly specialized vestibular nucleus Fibers of a secondary pathway arise from these nuclei and run to the medial longitudinal bundle of the same and opposite sides, where they divide into ascending and descending branches. Twigs are given off to the third, fourth, and sixth cranial nerve nuclei, and to the motor cells of the cervical spinal cord These twigs form reflex paths between the head, eyes, and vestibular apparatus

The caloric and rotation reactions are direct tests of the function of the vestibular apparatus. If a normal person with closed eyes and with his head erect be turned to the right about his vertical axis fifteen times in thirty seconds, upon stopping the rotation a

lateral nystagmus will be observed in both eyes. During the rotation the nystagmus will be in the direction of the rotation, but upon stopping the turning it is in the opposite direction, or in this case, to the left. Vertigo is also experienced, and when the rotation stops the patient still feels as if he were being turned. With eyes still closed, he points beyond an object in the direction of the turning and tends to fall to that direction. The head may be placed in various positions to bring the other canals into the plane of rotation, whereupon similar vertigo and nystagmic movements will occur. Similarly, in normal persons, syringing of the ear with cold water produces nystagmus toward the opposite side; if hot water is used, the nystagmus, on the contrary, is toward the side of the syringed ear.

If the vestibular nerve is blocked, and there is a "dead" labyrinth, nystagmus, vertigo, and past pointing will not occur. If there is an incomplete lesion of the nerve, there may be a delay in the entire response or a reduction in intensity. Practically, these tests are of corroborative value to determine the complete involvement of the vestibular nerve in acoustic neurinomas, but further than that their value in making a localizing diagnosis is problematical.

Glossopharyngeal, Vagus, Accessory, Hypoglossal.—The nuclei of these last four cranial nerves within the medulla are practically inseparable. As the ninth, tenth and eleventh nerves pass through the jugular foramen they are in close proximity to a point below the level of the tip of the mastoid bone. After the hypoglossal nerve emerges from the cranial cavity through the condyloid foramen, it follows the course of the three others in the retroparotid space. It can be easily understood how extracranial lesions, such as cervical adenopathies, tumors, infection of the jugular bulb, or acute adenitis of pharyngeal origin may involve these nerves singly or in groups.

The first accurate knowledge of the function of the glossopharyngeal nerve was obtained after it was divided intracranially for the relief of glossopharyngeal neuralgia. Previously, studies upon its function had been made following relatively gross injuries from tumors or trauma of the last four cranial nerves. There is no apparent demonstrable motor function of the ninth nerve since the pharyngeal muscles appear to be normal after its section, and there is no complaint of dysphagia or regurgitation of liquids. Although anatomists state that the stylopharyngeus muscle is supplied by the ninth cranial nerve, this paralysis cannot be demonstrated. There is a loss of sensation on the posterior third of the tongue, over the anterior, lateral and posterior walls of the pharynx from the lower nasopharynx to the epiglottis including its posterior aspect, over the tonsils, pillars, Eustachian orifices, and over a narrow rim along the front of the soft palate including the uvula.

It is difficult to conduct clinical tests for the function of the vagus nerve, and as one considers the point, it is amazing how few neurological signs are found in involvement of a nerve of such wide distribution and importance. If the entire trunk of the nerve is involved intracranially, or shortly after its exit, unilateral paralysis of the palate and larynx together with anesthesia of the larynx occur on the affected side. Hoarseness, due to paralysis of the vocal cord, and regurgitation of fluids through the nose due to paralysis of the soft palate occur. The latter may be observed by asking the patient to say "ah"; the median raphé will be pulled to the sound side. The projectile vomiting and slow pulse found in increased intracranial pressure are without doubt due to involvement of the vagus nerve which innervates the heart and gastro-intestinal tract.

The spinal accessory nerves innervate the sterno-cleido-mastoid muscles and weakness of those muscles may be found by having the patient turn his head to the right and left against the resistance of the examiner's hand. The trapezii muscles are also supplied by this nerve, and any weakness in their function may be determined by having the patient elevate his shoulders.

The effects of a lesion of the hypoglossal nerve may be easily demonstrated. When the tongue is protruded, it will deviate toward the side of the paralysis, and the atrophy and wrinkling upon the paralyzed side is striking. While the tongue lies within the mouth, it deviates toward the side opposite to the paralyzed nerve.

Several clinical syndromes have been described as a result of extracranial lesions involving these four nerves The *syndrome of Avellis* is characterized by a unilateral paralysis of the soft palate and vocal cord of the same side and is produced by a lesion of the internal branch of the spinal accessory and of the vagus nerves. A paralysis of the sterno-cleido-mastoid and trapezius muscles produced by a lesion of the external branch of the spinal accessory, in addition to the symptoms described by Avellis, characterizes the *syndrome of Schmidt* The presence of both of these syndromes plus a lesion of the hypoglossal nerve, which produces a unilateral paralysis of the tongue, gives rise to *Jackson's syndrome*. A lesion at the jugular foramen involving the ninth, tenth, and eleventh may also include the cervical sympathetic trunk In this case enophthalmos, narrowed palpebral fissure, and myosis are added to the clinical picture.

CEREBRUM

Our knowledge of localization within the cerebral cortex dates back to 1861 when Broca demonstrated that destruction of the left third frontal convolution may result in a loss of ability to speak

Nine years later Fritsch and Hitzig demonstrated that electric stimulation of the cortex in the region of the central sulcus will elicit movements from muscles of the opposite half of the body. Later physiological and pathological investigations have served to outline a number of such so-called centers with precision.

As the brain increases in size, the area of the cortex of gray matter expands out of proportion to the increase in volume of the white matter upon which it rests and is thrown into folds, or gyri, which are separated by fissures or sulci. Thus the frontal lobe is bounded below by the lateral cerebral (Sylvian) fissure and posteriorly by the central sulcus (Fissure of Rolando). The parietal, occipital, and temporal lobes have rather arbitrary boundary lines. The parieto-occipital fissure may be taken as the dividing sulcus, and an imaginary line which is a continuation of the lateral cerebral fissure separates the temporal from the parietal and occipital lobes. A part of the cerebral cortex which overlies the corpus striatum lags behind in its development and becomes overlapped by the surrounding cortex. This conical mass, called the Island of Reil, becomes hidden from view at the bottom of the lateral fissure. The cerebral hemispheres are joined by a large mass of white matter, the corpus callosum, which consists of bundles of association fiber tracts connecting the two halves of the brain.

The central nervous system of man is the result of the superimposition of more recently developed portions. In the lowest forms of organisms the simplest types of reflex movements exist, and as newer reflexes are added, these inhibit the older and simpler types. There has resulted a brainward shifting of control of many functions, the centers of which lie in more caudal parts of the nervous system in lower forms. This encephalization of function reaches its highest development in man. Function is spread over the cerebral hemispheres as one ascends the animal scale.

Frontal Lobe.—It has been the custom to speak of the frontal lobes as "silent" areas of the brain, but it must be remembered that the posterior boundary of the frontal lobe is the central sulcus, or the Fissure of Rolando and the inferior boundary is the lateral cerebral, or Sylvian, fissure. The motor projection centers are located in the anterior wall of the central sulcus, in the adjacent part of the anterior central gyrus and in that part of the cortex which lies rostral to the continuation of the central sulcus on the medial surface of the hemisphere. These motor areas are arranged in inverted order, beginning with the center for movements of the toes near the dorsal border of the hemisphere and ending with that for the face at the lower end of the gyrus. This area of the frontal lobe is termed the precentral motor cortex and it is the principal effector mechanism of the cerebral cortex by means of which the activity of the brain is expressed through the skeletal muscles.

The *precentral motor cortex* has been subdivided into areas, 4, 6, 8, and 44 according to Brodmann's scheme. (Plates II and III.) Area 4 lies just along the anterior lip of the central sulcus and from it the impulses which initiate voluntary movements on the oppo

side of the body descend to the motor nuclei of the cranial and spinal nerves. Movements of the toes are initiated in that part of the area nearest the dorsal border of the hemisphere while those for the cheeks, jaws and lips are represented at the ventral, or lower end of the area. Area 6 lies just rostral to area 4 and within it there is some localization of function, but nothing as discrete as that within area 4 Area 8 is not sharply defined from area 6 but lies rostral to it. Movements elicited from this area concern primarily the extrinsic muscles of the eye but it also contains an area within it, stimulation of which produces pupillary changes, lid movements and conjugate movement of the eyes. In man, area 44 has been elaborated into the speech area of Broca, but there is little known which differentiates it from the adjacent area which integrates the simpler motor functions of the eyes, tongue, lips and pharynx.

The precentral motor cortex is a complex cortical area in which the microscopic structure varies.

Area 4r is characterized by gigantic pyramidal cells in the fifth layer which give rise to the largest fibers in the pyramidal tract. Area 4a is identical in microscopic structure except for the absence of giant pyramidal cells. Area 4s contains a band of large pyramidal cells which are more superficial than the giant pyramidal cells in Area 4r. Area 6 resembles Area 4a except its cells have a tendency toward arrangement in columns. It gives rise to a large number of extrapyramidal fibers which descend to the thalamus, basal ganglia, red nucleus, and other subcortical centers.

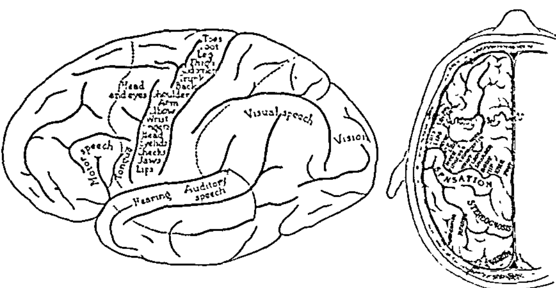
Areas 4 and 6 receive many afferent fibers from all other regions of the cerebral cortex, principally the sensory postcentral gyrus and thalamus.

Fibers from the precentral motor cortex are concerned with the co-ordination of visceral and vasomotor functions Just how this influence is exerted over the hypothalamus, which is the chief central mechanism for the control of these functions, is unknown Nevertheless, it influences the cardiac rate, blood pressure, dilatation or constriction of the vascular bed and alters gastrointestinal motility as well as lesser vegetative functions.

In their monograph, *The Isocortex of Man*, Bailey and Von Bonin state that large areas of the cerebral cortex are so similar in microscopic structure that any attempt to divide them is useless

That part of the frontal lobe anterior to the precentral motor cortex is often spoken of as the "silent" area of the brain, mainly because its involvement by disease does not produce a striking and dramatic clinical picture of paralysis. Yet, the symptoms produced by a tumor of the anterior portion of the frontal lobe are definite and valuable in diagnosis and may be classed on the whole as mental or psychic Their onset is insidious and may often be regarded only as temporary peculiarities, but later they may be appreciated as definite evidence of frontal lobe disturbance. The patient becomes disinterested in his surroundings, family and business

PLATE II



DIAGRAMS WHICH SHOW THE APPROXIMATE LOCALIZATIONS OF VARIOUS FUNCTIONS IN THE CEREBRAL CORTEX

He shows a complete reversal in his personal habits; if previously neat in appearance, circumspect in speech and inclined to worry over matters, he becomes slovenly in dress, dirty, obscene, facetious, and wholly unconcerned or careless about his personal affairs. Often such patients fail to show by word or deed any sign of interest when they are told of their physical state and its serious import.

These same symptoms may be found more pronounced in patients with a tumor of the corpus callosum, that large bundle of association fiber tracts which connects the two cerebral hemispheres. As a matter of fact, frontal lobe tumors often involve the corpus callosum because of its anatomical position.

Stupor and apathy may occur as the result of increased intracranial pressure which accompanies tumors located elsewhere and should not be confused with true frontal lobe mental symptoms. Reduction of intracranial pressure by the use of intravenous hypertonic solutions usually causes these symptoms to disappear. On the other hand, frontal lobe symptoms persist and may even stand out more clearly.

The initial clinical symptom in a lesion of area 4 immediately anterior to the Rolandic fissure, or central sulcus, may be a weakness, paralysis, or convulsive seizure of a part or all of the portions of the body represented. The severity of motor involvement of the face, arm, or leg depends upon the exact relation of the lesion to the precentral motor cortex. For example, a tumor situated anterior or posteriorly may involve area 4 by pressure, or it may produce direct destruction of the precentral gyrus. Many intracranial tumors produce irritative symptoms, such as convulsive seizures which may involve only the face, arm, or foot in their onset and then spread to involve the entire musculature of the same side. From these local beginnings, a generalized convulsion may occur. Very commonly however, such a spread does not occur and rapid clonic movements remain confined to a relatively small portion of the motor area. In such limited attacks (Jacksonian convulsion), consciousness may not be lost; but often a transitory postconvulsive weakness, or paralysis, occurs which aids greatly in making a localizing diagnosis, and may be the beginning of a permanent weakness. While convulsions occur frequently from causes other than an intracranial tumor, nevertheless an epileptiform seizure which occurs for the first time in an adult must be suspected as being due to a tumor. It is the duty of the doctor, and not the patient, to prove that a tumor is not present.

Often when the premotor area of the frontal lobe is involved gradually, generalized weakness of the contralateral extremities is manifested best by the patient's inability to perform skilled movements

with the fingers. With a gradually developing lesion in area 6, spasticity, with increased tendon reflexes, forced grasping and vasomotor disturbances may appear late. The grasp reflex is elicited by stimulating the skin of the palm of the hand with the handle of a reflex hammer, stroking distally. If present, a powerful flexion of the fingers occurs which the patient relaxes slowly and with difficulty.

Lesions of area 44 and the areas immediately about it may cause a defect in rapid and automatic performance in response to a command; that is, apraxia of the larynx, tongue and lips which makes it impossible for the patient to perform the movements of articulation, even though he may know exactly what he wishes to say.

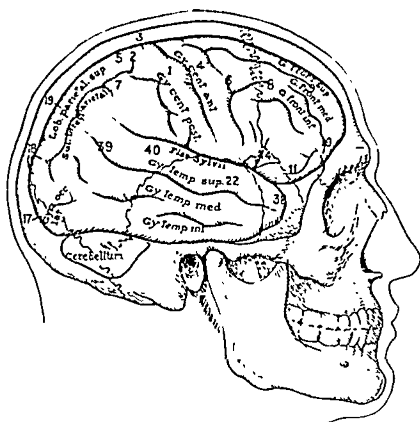
When area 8 is irritated, the eyes turn to the opposite side; occasionally the eyes may turn toward the opposite side and upward, and rarely downward. If the disturbance remains local, the head does not take part in the reaction. In a convulsion the eyes are turned by a series of clonic movements to the opposite side, then the movements become tonic. If this area is destroyed, the eyes may deviate toward the side of the lesion and the patient may be unable to move the eyes to the contralateral side.

Parietal Lobe.—The *sensory projection centers* of the cortex are those areas within which the sensory projection fibers from the periphery terminate. This common sensory area, in which general sensations from the surface of the body and the deeper tissues end, is located in the gyrus just posterior to the central sulcus The evidence for the location of these sensory areas was provided by Cushing's observations on the electrical excitability of this portion of the human cerebral cortex. There is no known topical representation of the body in this area.

A lesion which involves this area directly, or by contiguity, produces an unequal diminution in the various types of sensation, but rarely a complete loss. Rather, the patient loses his ability to localize a painful stimulus or to measure its intensity. He cannot determine the relative weights or size of objects; he might be unable to recognize a change in position passively imposed upon his extremities, and he cannot differentiate between the texture of materials. Neither can the patient recognize an object placed in his hand with his eyes closed. This inability to correlate and interpret various sensory impressions is known as astereognosis.

The optic radiations course through a portion of the parietal lobe to reach the visual center, and a deeply-placed tumor, for example, may produce a contralateral, homonymous visual field defect Usually this is a lower quadrantic defect and not a hemianopsia.

PLATE III



PROJECTION OF THE LOCATION OF THE IMPORTANT GYRI ONTO THE SURFACE OF THE SKULL.

The figures indicate the location of certain areas of Brodmann on the cerebral cortex.



Temporal Lobe.– The *olfactory receptive center* is located in the uncus and adjacent portions of the hippocampal gyrus of the temporal lobe and the *auditory receptive center* is situated in the anterior transverse temporal gyrus, which lies buried in the floor of the lateral sulcus (Sylvian fissure). This latter area receives the auditory radiation from the medial geniculate body. In addition, the optic radiations sweep backward through the temporal lobe to reach the visual receptive center.

Lesions which affect the uncinate gyrus give rise to indefinite unpleasant olfactory auræ, usually followed by a dreamy state which lasts a few seconds. Such attacks have been termed "uncinate gyrus fits." At the onset, the patient usually smacks and licks his lips, stares into space, and loses contact with his surroundings. The attack is usually described as a dream in which the patient's strange surroundings seemed very familiar.

A tumor in the temporal lobe may in its expansion produce pressure upon the precentral motor cortex of the frontal lobe. As a result, facial weakness or even involvement of an arm or leg may occur.

If the optic radiations are involved, and they commonly are unless the tumor is small or located far anteriorly or inferiorly, a homonymous hemianopsia is produced. Visual hallucinations are also common and they are characteristic in that they are of typically formed people, animals, buildings, or other objects.

Occipital Lobe.—The *visual receptive center* is located in the cortex which forms the walls of the calcarine fissure and in the adjacent portions of the cuneus and lingual gyrus in the occipital lobe. The fibers of the optic radiation from the pulvinar and lateral geniculate body terminate in the visual projection center. These fibers carry impulses from the temporal side of the corresponding retina and the nasal side of the opposite one. Consequently, the visual cortex of one hemisphere receives impressions from objects on the opposite side of the line of vision.

A destructive lesion of the occipital lobe produces a contralateral homonymous hemianopsia. The differentiating point between the hemianopsia produced by an occipital lobe lesion and one in the temporal lobe is that in the former the macular region is spared, whereas in the temporal lobe lesion the hemianopsia passes directly through the point of fixation.

Visual hallucinations which occur as the result of irritation of the occipital lobe are described by patients as flashes of light, brilliant stars, a rainbow of colors or bright lines, in contrast to the visual hallucinations of form present in irritative lesions of the temporal lobe.

Obviously the sensory and motor projection centers and the special sensory receptive centers occupy only a small part of the entire area of the cerebral cortex. The remaining portions are connected with these centers by association fibers and are known as association centers. They are of especial significance in the development of the higher intellectual functions of man. It is wise to be very conservative in locating any mental faculty or fraction of conscious experience in any part of the cerebral cortex.

Aphasia.—An idea of the significance of the association centers may be obtained from a study of speech defects which result from lesions of the cortex and are included under the term *aphasia*. In right-handed individuals these result from lesions in the left hemisphere. Destruction of the triangular and opercular portions of the inferior frontal gyrus usually causes loss of ability to carry out the coördinated movements required in speaking, but it does not impair the ability to move the tongue or lips. This defect is known as motor aphasia. Likewise, after a lesion in the posterior part of the left superior temporal gyrus the patient may hear the spoken word but no longer is able to comprehend its meaning. This is sometimes known as sensory aphasia or word deafness. Word blindness, the inability to understand the printed or written word although there is no impairment of vision, may result from lesions in the angular gyrus. It is not as yet altogether clear to what extent such lesions are dependent upon the destruction of association tracts which lie subjacent to these centers. In fact Head was of the opinion that although speech may be affected by the destruction of brain substance, the faculty of speech is not located in any particular cortical area. It was his belief that no lesion, no matter how sharply localized, can affect speech alone or produce real motor or sensory aphasia; in other words, in every case there is an involvement of all the elements of speech

Certain tests should be employed to determine the presence or absence of disorders of speech in a unilateral lesion of the brain. The patient should be examined for his ability to name and recognize objects and colors. He should be tested for his power of reading and writing in their simplest form; that is, by the use of words consisting of three letters only. He should be required to perform acts by imitation and upon written and spoken commands.

According to Head, patients suffering from disorders of speech are able to do anything which does not require symbolization. Since words are the commonest and most obvious symbols used in thinking, all acts suffer which demand any form of verbalization for their execution In other words, symbolic thinking and expression are

affected, and the more acute and severe the lesion, the graver the disorder. Dissociated manifestations of such disorders produced by a given lesion gives rise to the various types of aphasia. For example, a patient cannot read to himself because he cannot retain a long series of words. His enunciation is slow, and his vocabulary is greatly restricted, often being reduced to "yes" and "no." Writing shows the same defect as articulatory speech, but improves as verbalization returns. Such patients usually recognize the errors committed and are said to suffer from *verbal aphasia*. *Nominal* aphasia consists in the defective use of names. Reading is difficult, particularly if an attempt is made to spell out the words. In *syntactical* aphasia the patient talks jargon. Articulation and phrasing are defective and poorly balanced. *Semantic* aphasia produces a want of recognition in the full significance of words and phrases. Details may be very easily grasped, but the full meaning is lost.[1]

Subcortex and Basal Ganglia.—The projection fibers from the motor cortex converge within the internal capsule as they continue downward into the brain stem and spinal cord. A small lesion in the internal capsule may produce a widespread paralysis which involves the face, arm, trunk, and leg. If the hemiplegia is associated with a hemianesthesia and a hemianopsia, the site of the lesion is in the posterior limb of the internal capsule where the motor, sensory, and optic nerve fibers lie side by side.

In close proximity to the internal capsule lie the basal ganglia, consisting essentially of the *caudate* and *lenticular nuclei*. The caudate nucleus and the *putamen* (outer segment of the lenticular nucleus) constitute a newer structure, the neo-striatum or *striatum*, while the remaining portion of the lenticular nucleus, the *globus pallidus*, is called the paleo-striatum or merely *pallidum*. Some include the red nucleus, the substantia nigra, and other structures in functional relationship to the basal ganglia. The strio-rubrospinal system is older than the corticospinal, and in accordance with the concept of encephalization, the latter inhibits the former. Both pathways inhibit tone, and when both are destroyed as in a large subcortical lesion, hypertonus is extreme. By interaction with area 6 of the precentral motor cortex, the basal ganglia coördinate and smooth out voluntary motor acts integrated through the motor cortex.

Chief among the abnormal states are the syndromes of *parkinsonism* (paralysis agitans) and various involuntary movements which

[1] Reference should be made to Nielsen's contributions which are of great aid in understanding the disturbances of language. Nielsen, J. M., "Agnosia, apraxia, aphasia; their value in cerebral localization," Bull Los Angeles Neurological Society, 1936, vii, 210, "Textbook of Clinical Neurology," Paul B Hoeber, Inc

include *tics*, *tremor spasms*, *myoclonus*, *palilalia*, *athetosis*, and *dystonia*. In parkinsonism there is a loss in facility of movement, slowness, rigidity, with a defect in associated movements such as arm-swinging. The face has a stolid masked expression. The tremor is often of the pill-rolling type. The rigidity is cog-wheel in type, and the body has a flexion posture. Occasionally pulsions in any direction are seen. The spasms may be masticatory, oculogyric, or of other types. In an oculogyric crisis, the eyeballs may be held for hours in upward or downward gaze. Partial ablation of the precentral motor cortex in man has been shown to subdue the involuntary movements which result from disorders of the basal ganglia.

Thalamus and Hypothalamus.—The *thalamus* is the great subcortical sensory station connecting afferent impulses with the cerebral cortex, chiefly in the parietal region, and also the station for instinctive forces grouped loosely under the term "emotions" like hate, fear, anger, and love. The so-called thalamic syndrome consists of contralateral loss or diminution of sensation, especially deep, and agonizing, burning pain which is intractable. One limb, one-half of the face, or the entire half of the body may be affected. Sensory stimuli barely perceivable evoke an excessive unpleasant response on the affected side. The point stimulated may not be localizable by the patient.

The *hypothalamus* consists of the *pars optica*, the *pars mamillaris*, and the *subthalamus*. The pars mamillaris includes the mamillary bodies, the tuber cinereum, the infundibulum, and the hypophysis. The subthalamus forms a zone of transition between the thalamus and the tegmentum of the mesencephalon. Water metabolism is governed by centers lying in the tuber cinereum, and when this part is involved by tumor, trauma, or inflammation, polyuria and polydipsia result. Diabetes insipidus may occur from a lesion in this area. The hypothalamus also plays a rôle in the control of sugar metabolism, fat metabolism, sex function, temperature regulation, ocular sympathetic control, and sleep regulation. The nearness of the hypophysis and its integration into the hypothalamic mechanism results in varied defects of the above mentioned functions when this gland is involved by neoplastic change. The classical hypothalamic syndrome is rarely a clear-cut condition in man because of concomitant pathology in one or more of the endocrine glands (frequently the hypophysis) and various resultant alterations in the autonomic nervous system, which is largely dependent for its normal functioning on the hypothalamus and endocrine system.

Brain Stem.—Lesions of the brain stem are more often vascular and inflammatory than neoplastic or degenerative. Due to the compactness of major neural elements in these regions, small lesions

produce major signs. The residual syndromes often show combinations of cranial nerve involvement on the same side and pyramidal tract and/or sensory tract defects on the contralateral side.[1] The combination of homolateral cranial nerve and contralateral pyramidal tract signs points to a lesion of the brain stem, the level being that at which the cranial nerve is affected.

CEREBELLUM

The cerebellum is composed of three parts: the *vermis*, is small, unpaired, and lies in the middle of two large lateral masses; the *cerebellar hemispheres*, which are connected with each other by the vermis.

Early investigations by Fluorens and Luciani developed the concept that the cerebellum functions as a whole. Support for this unitarian theory came from the anatomical research of Golgi, Cajal and Sherrington. As a result of his studies of the influence of the cerebellum on muscular coordination, the latter introduced the well known phrase, "head ganglion of the proprioceptive system." A dissenter from this viewpoint was Bolk who maintained that there was functional localization in the cerebellum. Both groups agreed, however, that the primary function of the cerebellum was the coordination of muscular activity, with particular emphasis on afferent connections with proprioceptor endings.

The most recent experiments on electrical activity of the cerebellum by Moruzzi, Snider,[2] Clark and others have established certain facts. Tactile, auditory and visual areas exist within the cerebellum. Motor, tactile, auditory and visual areas in the cerebrum project to these same cerebellar areas, which project back on to the cerebral areas. In fact, almost every structure which projects to the cerebellum receives a projection from it though it is seldom a direct one. It has been shown that stimulation of the cerebellum can modify the activity of the cerebrum and certain areas, when stimulated properly, produce localized movements and suppression or facilitation of cortically or reflexly induced movements.

Thus, it would appear necessary to abandon the older concepts of the cerebellum functioning as a whole and entirely in a proprioceptive sphere. Rather, the cerebellum acts from localizable areas to dampen and potentiate associated sensory and motor centers.

[1] The long tracts, like the pyramidal or the lemnisci, cross below the level of the uncrossed cranial nerves.

[2] Snider, Ray S. Recent Contributions to the Anatomy and Physiology of the Cerebellum, Arch Neurol. and Psych., 64, 196, 1950.

Cerebellar disease produces homolateral hypotonia, ataxia, incoordination, or asynergia, and weakness. The normal smooth movement is replaced by a jerky, misdirected, poorly-measured one. This has been called "decomposition of movement." Other signs of cerebellar dysfunction may be explained by presence of hypotonia and dyssynergia. The former produces the flail-limb with increased mobility at the joints and defective after-shortening of muscles. This often results in a pendular swinging of the leg after elicitation of the knee jerk. Dyssynergia produces misdirection, over- or undershooting the mark, loss of check reflexes, ataxia, and faulty alternate contraction and relaxation of muscles (adiadokocinesis).

If it became necessary to reduce the symptoms of loss of cerebellar function to the minimum, one could not do better than to use the words *dysmetria* and *asynergia*, the difficulty of measuring movements, and the lack of ability to coördinate movements.

Tumors of the cerebellum may involve primarily the vermis or the lateral cerebellar lobes. Neoplasms of the vermis, and they are most common in children, may be accompanied only by a profound trunkal ataxia, hypotonia of the muscles, and a serious difficulty in walking. The gait is very uncertain, the base is wide, and the patient tends to fall backward.

Neoplasms of the lateral lobe, on the contrary, are accompanied by more symptoms. The patient complains of a suboccipital headache and his neck muscles are tender. The occiput is rotated and the head is tilted toward the affected side. Attempts to flex the head on the chest are painful and are resisted. The muscles are hypotonic and the wrists and fingers can be hyperextended. If the forearm is shaken, the hand flaps about. When the forearms are flexed against passive resistance and this is suddenly released, the hand may strike the patient's body or face because of his inability to check rapidly a movement once initiated. Alternating movements of the fingers or hands cannot be performed smoothly and rapidly; the hands and fingers are awkward and clumsy. The fine movements which bring out this lack of coördination and control in the upper extremities cannot be used in testing the legs, but the finger to nose test can be duplicated by the heel to knee test, both of which bring out dysmetria.

Disturbances in gait and station are particularly striking in tumors of the lateral cerebellar lobes. The patient stands and walks with his feet wide apart. He sways from side to side as though intoxicated in an effort to maintain his balance and has a tendency to fall toward the side of the lesion.

Nystagmus is a frequent symptom of a tumor in the lateral lobes, but may be entirely absent in a neoplasm restricted to the vermis.

Fixation is essential to cerebellar nystagmus and the movements are slower and coarser on looking toward the side of the lesion. The ataxic movements of the eyeballs are comparable to the dysmetric terminal movements of the extremities.

Tumors of the cerebellum may produce symptoms due to pressure upon neighboring structures, particularly the cranial nerves in the cerebello-pontile angle. Loss of the corneal reflex, weakness of the face, and difficulty in swallowing are frequently present.

Increased intracranial pressure is present very early and more frequently than in tumors elsewhere. The cerebrospinal fluid pathway becomes obstructed by pressure from the tumor and a secondary internal hydrocephalus quickly develops. Papilledema develops rapidly and very often one or both abducens nerves are paralyzed as the intracranial pressure increases.

CHAPTER II

CRANIOCEREBRAL INJURIES

"The difficulties connected with this part of surgery are sufficiently proved by this circumstance, that, notwithstanding it has at all times excited the attention of surgeons of the greatest talents, and possessing the most extensive field for observation, much difference of opinion still subsists, and the practice that ought to be followed in particular cases yet remains a matter of dispute."

JOHN ABERNETHY.

CRANIOCEREBRAL injuries are no longer a problem whose solution is reserved for the neurological or general surgeon. Every physician is often confronted with the patient who has been seriously injured in an automobile accident. These experiences should have taught him that there can be no advocacy of one method of therapy in all injuries to the skull which are accompanied by cerebral trauma. It has been learned that many patients so injured never reach the doctor; that in many instances a judicious minimization of active therapy is the best treatment; that the intravenous administration of hypertonic solutions and properly indicated lumbar punctures may be life-saving measures; that the operation of subtemporal decompression, so strenuously advocated in the past for the treatment of these patients, is followed by a fatality more often than by recovery in inexperienced neurosurgical hands and finally, that in many cases the injury is so overwhelming that from the beginning the most heroic measures are of no avail.

Fractures of the skull are unique among the fractures of other bones of the body in that the fracture is relatively unimportant. The injury to the brain should receive primary consideration. This trite statement has been made very often and under many disguises but cannot be overemphasized because it is so often forgotten when the doctor is constantly urged for a statement as to whether or not a fracture of the skull is present. It is only of academic interest to classify or describe in detail the various fractures of the cranial vault or base. Likewise, classifications of symptoms, which cause the physician to speak glibly of "concussion" or "contusion," and influence him to fit his case into a given descriptive mold lead the inexperienced to institute a set régime of therapy and do not emphasize the fact that each case of skull injury is an individual problem to be treated, if necessary, by a combination of methods. There can be no hard and fast division of the clinical symptoms or the pathological changes which accompany craniocerebral injuries

ARTERIAL, VENOUS AND CEREBROSPINAL FLUID CIRCULATION

To understand the clinical symptoms adequately it is necessary to recall some of the fundamental facts of the mechanism of the arterial, venous and cerebrospinal fluid circulation so intimately concerned with craniocerebral injuries.

The arterial circle of Willis is formed by the two vertebral, the two internal carotid arteries and six branches which reach the surface of the cerebral hemispheres where they spread out in the pia arachnoid. During their course, they supply the basal ganglia, the choroid plexuses and to a small extent the white matter. From the surface, innumerable arterioles dip into the cortex and subcortical white matter where they become smaller and smaller and finally form a capillary bed. The arterioles and capillaries of the brain anastomose freely with each other and also with branches of deep arteries which come up through the white matter. It has become known, therefore, that there is a rich collateral arterial circulation in the brain. The arterial capillaries are similar to those elsewhere in the body, but they are less subject to local vasomotor control than are those found extracranially.

The venous circulation of the brain is less regular; there are more anastomoses, and the veins are more numerous. One group collects blood from the choroid plexuses, the ventricles and the basal ganglia and empties into the vein of Galen and thence the jugular bulb. The second system drains blood from the subcortex and cortex into vessels which lie in the pia mater on the surface of the cerebral hemispheres which in turn empty into the large dural sinuses. These veins leave the cerebral surface at a right angle, perforate the pia mater and arachnoidea, course within the dura mater, and then enter the sinus. This anatomical relation favors their laceration in cranial injuries.

There are definitely discernible spaces about the large and smaller arteries and veins in the nervous tissue and potential spaces about the arterioles, venules and capillaries. In the cellular layers of the cortex, the capillaries are numerous and run close to the individual nerve cells There is extracellular tissue fluid which lies in the pericellular spaces, from which it escapes into the pericapillary spaces; finally, reaching the perivascular and subarachnoid spaces This pericellular and perivascular fluid comes from the blood plasma in the capillary loops as it does in other organs analogous to the brain. As is also true elsewhere its circulation is dependent upon (1) intracapillary pressure, (2) the osmotic pressures of the blood plasma and the tissue fluids, and (3) upon the permeability of the capillary walls.

The cerebrospinal fluid which is produced largely by the choroid plexuses is poured directly into the lateral cerebral ventricles which are lined by ependymal cells. That portion of the fluid which is formed by the choroid plexus in the lateral ventricles passes through the foramen of Monro into the third ventricle, and then by way of the aqueduct of Sylvius into the fourth ventricle. From there the fluid passes into the subarachnoid spaces through the two lateral foramina of Luschka and the medial foramen of Magendie. From the dilatation of the subarachnoid space in the midline between the cerebellum and the medulla (cisterna magna) the fluid flows slowly downward into the spinal subarachnoid space. However, at the same

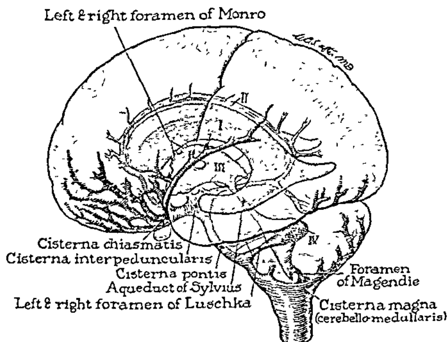


FIG. 40 — Diagram of the ventricles of the brain and the basilar subarachnoid spaces. (Courtesy of Dr Paul B Magnuson)

time it passes upward more rapidly about the base of the brain where other subarachnoid cisterns are present, and then more slowly over the cerebral hemispheres. This movement of the fluid is facilitated by impulses transmitted to it by the vascular system. According to the present anatomical descriptions, the subarachnoid space in which the fluid circulates is between the arachnoidea and the pia mater Numerous delicate spider web-like trabeculæ project from the arachnoidea to the pia mater. Flat, polygonal mesothelial cells cover the inner surface of the arachnoid, the trabeculæ, the surface of the brain, and all blood-vessels which pass through the subarachnoid space These mesothelial cells establish a periadventitial fluid channel about each blood-vessel which penetrates the nervous

system. The subdural space has but a slight relationship to the circulation of cerebrospinal fluid. However, the dura mater and the arachnoidea fuse at the points where the arachnoid penetrates the dense fibrous tissue of the dura mater. These are the arachnoid villi, by which the mesothelial cells of the arachnoid come directly beneath the vascular endothelium of the large dural venous sinuses. (Fig. 40.)

The cerebrospinal fluid then circulates everywhere about the central nervous system, in the ventricles, and in the meshes of the subarach-

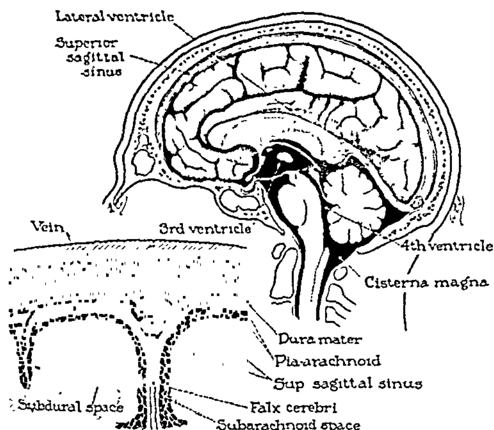


FIG 41.—Diagram showing the relation of the subarachnoid spaces, of the subdural spaces and the cranial venous sinuses (Courtesy of Dr. Paul B Magnuson.)

noid space. These channels are lined by special fluid-retaining cells so that a true circulation is maintained. In the arachnoid villi the fluid comes into close relation with the large venous sinuses of the dura mater, and it is at this point that absorption occurs. The mechanism of passage of the fluid is a process of filtration from a point of higher pressure to a point of lower pressure with later experimental evidence to show that the fluid is a dialysate in osmotic and hydrostatic equilibrium with the blood. There is a second method of absorption in which the fluid escapes slowly into the true lymphatic vessels in an indirect manner. (Fig. 41.)

A variation in the brain volume, circulation of the blood or cerebrospinal fluid must be compensated for in one or both of the other mechanisms within the skull. This is the principle stated in the Monro-Kellie law, and it serves as the basis for an understanding of the pathological physiology involved in craniocerebral injuries. Since the skull and vertebral column are relatively inelastic and rigid containers, the pressure of the cerebrospinal fluid and intracranial vascular pressures have a close relationship. It may be stated that the cerebrospinal fluid pressure varies with the blood-pressure, but follows more accurately the venous than the arterial pressure.

When the brain is injured, a reflex dilatation of the smaller cerebral arteries occurs, and there is a rise in intracapillary pressure due to this loss of check by the normal arteriolar contractility.[1] If this situation persists, the veins become engorged and capillary and venule pressures rise. Blood plasma moves from the arterial side of the capillary loops into the pericellular and perivascular spaces; absorption of tissue fluids into the venous side decreases, and the perivascular spaces become overfilled and *edema* occurs. In turn, edema further slows the circulation and tissue anoxemia occurs, and the vicious circle repeats itself. Eventually, the pressure in the large veins and dural sinuses increases, and cerebrospinal fluid cannot be absorbed. The therapeutic problem is, therefore, to help re-establish the normal fluid and vascular pressures, and to reduce the increased brain volume brought about by edema.

MECHANICS OF CRANIOCEREBRAL INJURIES

Investigations[2,3] have emphasized the facts that nerve tissue, blood, and cerebrospinal fluid have about the same density as water; that the brain substance does not change its size appreciably when subjected to a hydrostatic pressure, and that the brain offers very little resistance to changes in shape as compared to the resistance it presents to changes in size. Obviously, the rigidity of the skull is enormous compared to that of the brain.

Deformation of the skull has been recorded electrically and is, as would be expected, most severe near the point of impact where there is compression distortion. Simultaneously on the opposite side, there

[1] Fremont Smith, F., and Kubie, L. S The Relation of Vascular Hydrostatic Pressure and Osmotic Pressure to the Cerebrospinal Fluid Pressure, Proc Assn for Res. in Nerv and Ment. Dis, Baltimore, Williams & Wilkins Company, 8, 114, 1929

[2] Gurdjian, E. S and Lissner, H R J. Neurosurg, November, 1944, 1, 393–399, 1944 "Mechanism of Head Injury as Studied by the Cathode Ray Oscilloscope Preliminary Report." Gurdjian, E S Deformation of the Skull in Head Injury a study with the "Stresscoat" technique Surg Gyn & Obs., 81, 679, 1945, 83 219 1946, 85, 195, 1947

[3] Holbourn A H S Lancet, Oct. 9th, 1943, 438 "Mechanics of Head Injuries."

is tensile deformation. Since the brain substance is extremely incompressible and yet easily altered in shape, its constituents are pulled apart in proportion to the degree of a sliding type of deformation.

Another mechanism acts to injure the brain due to the changes in velocity of the head produced by the blow. Linear and/or rotational acceleration forces may be set up and of these, the latter are the most injurious. The small amount of motion allowed the brain in its closed skull box is possible only because of the to-and-fro movement of the cerebrospinal fluid. When the head is subjected to rotational acceleration forces, the brain lags behind and a type of shearing strain results. Denny-Brown and Russell[1] found it difficult to produce concussion in animals if the head was prevented from moving when it was struck.

The inferior surfaces of the frontal and temporal lobes of the brain are frequently lacerated in severe injuries. The relation of the ridge of the lesser wing of the sphenoid bone to the temporal lobes gives a good grip on the brain and makes it possible for the skull to rotate the brain. In other areas, the brain slips beneath the skull and less strain results. Because of the incompressibility of the brain, no spaces can be formed between the skull and brain and therefore, the brain can only slide along the interior surface of the skull Since the dura mater is firmly attached to the skull its movement is negligible. Therefore, the pia mater has a slight sliding movement in relation to the arachnoid and the latter, a greater movement in relation to the dura mater. In this process, the vessels which drain the cortical veins into the venous sinuses will be stretched, and may rupture at any point along their length, thus causing subdural or subarachnoid hemorrhages. In general, it would appear that the so-called contrecoup injuries are in reality rotational injuries of the brain.

CLINICAL SYMPTOMS

The most simple type of craniocerebral injury encountered is that in which the patient has received a blow upon the head, either by a fall or by being struck by a moving object. The period of loss of consciousness, which immediately ensues, may last from a few minutes to several hours; the blood-pressure falls, the respirations become shallow and pallor occurs. There may or may not be a laceration of the scalp, and a linear fracture of the vault of the skull may or may not be present. In any event, the period of loss of consciousness is the best direct indication of the severity of the

[1] Denny-Brown, D., and Russell, W. R.: Jour. Physiol., 99, 153, 1940, "Experimental Cerebral Concussion "

cerebral trauma, provided that a careful neurological examination has failed to reveal a disturbance of motor function or other symptoms attributable to direct cortical damage. If the term "concussion" is to be used, it should be reserved for this group of patients, many of whom never reach a hospital or a doctor. The condition must be differentiated from all cerebral injuries which are severe enough to produce multiple gross or microscopic hemorrhages, edema, or lacerations. Pathologically, only a mechanical derangement of the molecules within the nerve cells may occur in the mildest type of trauma to the brain resulting in loss of consciousness. Varying in degree, and to be judged clinically by the degree of loss of consciousness, swelling and liquefaction of the ganglion cells and their processes with the phenomena of satellitosis and neuronophagia and swelling of the myelin may occur in the deep layers of the cortex. It must be emphasized, however, that any degenerative changes which occur are not accompanied by gross or microscopic hemorrhages and, therefore, recovery of function may be prompt and complete.

In a second group of cases, in which the etiological factors may have been exactly the same, but more severe in degree, there may be a prolonged period of unconsciousness which alternates with periods of delirium and is finally succeeded by coma with a generalized flaccid musculature, respiratory arrhythmia, circulatory collapse, and death. Between these two arbitrary divisions, the symptoms of the majority of patients with craniocerebral injuries range. Hemorrhages, both gross and microscopic, necrosis, broken myelin, tumefied, tortuous nerve fibers or destroyed axones, granular ganglion cells with pyknotic and displaced nuclei or cells completely disintegrated characterize the pathological changes found in patients with severe damage to the brain. Any attempt to classify the pathological changes which occur in the brain must be an arbitrary one because they overlap and merge one into the other.

When the brain is severely lacerated, surgical shock is practically always present; disorientation, confusion, and delirium may be extreme, or a deep coma with generalized muscular flaccidity may be present. Bleeding and a discharge of cerebrospinal fluid from the ears or nose, and extensive conjunctival and periorbital hemorrhages may be present. Blood may not escape from the ear and yet the tympanic membrane may be bluish-red and bulging from a hemorrhage which has not perforated through the drum. It may be concluded that under such circumstances blood has found its way into the subarachnoid spaces. As a result of the blood in the subarachnoid spaces, the patient may be extremely restless and difficult to control In addition, there is always a variable degree of rigidity of the neck which is indicative of meningeal irritation

One or more of the cranial nerves may be damaged in severe cerebral injuries. Anosmia, due to contusions of the under surface of the frontal lobes and damage of one or both olfactory bulbs is commonly encountered. Fractures through the orbital foramen may compress or sever the optic nerve, or the optic chiasm may be lacerated, usually in the antero-posterior diameter, to produce a bitemporal hemianopsia. The oculomotor, trochlear and abducens nerves may be injured independently, but often a total ophthalmoplegia with ipsilateral blindness, the result of a fracture through the orbital fissure and optic foramen, occurs. Facial paralysis due to injury of the terminal fibers of the seventh nerve in the facial muscles or to severance of the facial nerve in its canal is not uncommon. The acoustic nerve may be affected by a fracture or by hemorrhage and varying degrees of deafness with tinnitus may result. Injury of the vestibular portion of the eighth cranial nerve may produce hypersensitivity of the labyrinth, characterized by ataxia, nystagmus and severe vertigo upon sudden movements. Injuries to the last four cranial nerves are usually the result of penetrating injuries of the skull.

Respiratory arrhythmias, a slow pulse and high pulse-pressure, and a rise in temperature may be followed quickly by fixed dilated pupils, a sign of impending death. In the event of recovery the return of consciousness is slow, headaches are severe, and there may be a long period of amnesia. Naturally, in the presence of such severe cortical injury, brain edema and increased intracranial pressure remain high. The cerebrospinal fluid, at first very bloody, changes later to a brownish yellow and then to a clear yellow fluid. This color often remains for a long period even after brain volume and intracranial pressure have returned to normal.

In still another group of patients, depression of the fractured fragments of the skull may be present. The inner or outer table alone may be depressed, or there may be a complete solution of continuity so that both tables rest upon the underlying brain. Depressed fractures of the skull may be present with or without a laceration of the scalp. In severe crushing injuries, the scalp may be lacerated severely and the skull fracture may not only be comminuted, but some of the fragments may be driven into the brain. In such instances there are tears in the dura mater and destruction of brain tissue, which may be so pulpified as to escape from the wound.

In contradistinction to the clinical symptoms just described in which coma immediately follows the injury are those cases in which coma appears some time after the injury. There may be a short period of loss of consciousness followed by a lucid interval with the onset of a second period of loss of consciousness. Or, the patient

may have had no loss of consciousness immediately after the injury and then later gradually becomes comatose. This group of symptoms will be recognized as those characteristic of the classical picture of hemorrhage from the middle meningeal artery. Many descriptions have been given of the combination of symptoms involving the pulse, blood-pressure, and respirations under such circumstances. However, the most pathognomonic sign of middle meningeal hemorrhage is *dilatation of the pupil upon the side of the hemorrhage.*[1] It should be remembered that bleeding from the middle meningeal artery may occur on the side opposite to that of the skull injury. The importance of this index of increasing cerebral compression cannot be insisted upon too strongly. If the bleeding occurs slowly, and it is always extradural in these cases, the ensuing coma is slow in developing so that early the patient may be only stuporous Under such circumstances, a careful neurological examination will reveal a weakness of the muscles of the face and arm certainly, and possibly the leg. To request a patient who is stuporous to grasp or to perform gross voluntary movements which require considerable effort is not an accurate method of determining slight differences in muscle strength. One should observe the facial muscles in emotional expressions; the differences in the size of the palpebral fissures, or the tendency of the weak upper extremity to fall away as both extremities are held outstretched. It is such slight but definite evidences of motor weakness which corroborate the surmise of an increasing lesion over the opposite side of the cerebral cortex.

Even though the patient loses consciousness rapidly and cannot coöperate in the examination, one may, by careful observation, detect a difference in the tone of the muscles upon the two sides of the body. The upper eyelids may be raised passively and allowed to fall. On the affected side the eyelid may cover the eyeball slowly or not at all, while on the normal side it quickly returns to its original position. Likewise, the arm may be raised over the face or chest of the patient and allowed to fall. Though the patient may not voluntarily move either extremity, the difference in muscle tone may be observed quite readily as the normal arm avoids the face in its descent Passive flexion of the lower extremities followed by sudden release is a valuable method of eliciting a difference in muscle tone in the legs. The paretic limb *falls quickly and in an abducted* position; whereas, the normal limb gradually slides into its original position

Hemorrhage beneath the dura in an amount sufficient to produce

[1] This symptom is not always present at the time of examination because the size of the pupil changes frequently. When it is observed, it is a valuable diagnostic sign.

symptoms may occur relatively soon after a skull injury, or many months or years may elapse before the gradually developing hematoma produces symptoms. Subdural hematomas are not uncommon, and yet, because of the gradual onset of symptoms, they are not easily diagnosed. The same methods for the detection of slight differences in muscle tone on the two sides of the body may be utilized in these cases to great advantage. Many cases of chronic subdural hematomas are upon record which have produced symptoms years after a supposedly trifling skull injury.

The interval syndrome just described may also be produced by gross contusions of the temporal lobe, traumatic intracerebral hemorrhage, traumatic cerebral softening, subdural fluid accumulations and abscesses. When a large contusion of the temporal lobe has occurred there may be only a partial recovery from the initial deeply comatose state. The relapse into coma in these conditions is due to increasing intracranial pressure due to an accumulation of extradural, subdural or intracerebral blood, to edema of the temporal lobe, to a progressive subdural cerebrospinal fluid accumulation or to softening and swelling of the brain following arterial thrombosis.

The symptom complex of decerebrate rigidity may be produced by the edema which follows severe injury to one or both temporal lobes or to direct injury by severe basal fractures, collections of blood in the basal subarachnoid cisterns, or hemorrhages in the midbrain and upper pons. Microscopic hemorrhages in the hypothalamus may produce hyperthermia and in the event of recovery, diabetes insipidus, glycosuria and somnolence.

Often the patient with a craniocerebral injury may be physically restless, even in the presence of stupor. He may turn from side to side, constantly disarranging the bed clothing, is uncoöperative and resistant. These symptoms are most commonly caused by blood in the cerebrospinal fluid, but there is undoubtedly an associated multiple and diffuse contusion of the cerebral cortex, though not severe, which accounts for the cerebral symptoms.

Finally, several complications may follow skull injuries which add to the gravity of the patient's prognosis. Every patient who has a discharge of cerebrospinal fluid from the ears or nose is potentially a patient in whom a suppurative meningitis may develop. An apparently simple linear fracture of the skull in which a laceration of the scalp has occured may be followed by the development of an intracranial abscess accompanied by neurological symptoms dependent in their character upon the location and the chronicity of the infectious process. There are many cases upon record in which air has been found within the cranial cavity, or within the cerebrum, upon

roentgen-ray examination of the skull. These cases of penumocephalus follow (1) fractures through the accessory nasal sinuses which extend into the cranial cavity, and are often associated with a discharge of cerebrospinal fluid from the nose; (2) fractures through the petrous bone and the middle ear, and (3) compound fractures of the occipital bone with penetration of the posterior horn of the lateral ventricle. The air may be found in the subdural space, the subarachnoid system, within the brain substance or within the ventricles.

It should not be assumed that the changes in the pulse, blood-pressure, and respirations which accompany skull injuries are not of importance. However, they fluctuate tremendously in individuals *who may have similar types of injuries, and what is more important* they may vary markedly within short periods of time in the same individual. Therefore, while a careful, frequent record of the pulse, blood-pressure, and respirations should be kept, their greatest value lies in the evidence they provide of the effects of therapy rather than of the aid they give in establishing a diagnosis. It is not uncommon to be misled by a slow pulse in an individual who normally has a bradycardia.

It is to be emphasized again that each patient cannot be filed in any one category of symptoms. A middle meningeal hemorrhage, or subdural hematoma, may develop in an individual who received an apparently minor injury to the skull. On the contrary, a compound, comminuted fracture of the skull with depression of the fragments may never suffer a loss of consciousness. Likewise, a patient with an extensive laceration of the scalp received from a blow of comparatively small force may be in shock from a loss of blood. The important point then is to examine each patient carefully for any evidence of cerebral damage; to be alert to the possible onset of symptoms which are significant, and finally, to recognize the principles underlying their treatment and the methods which can be employed effectively to aid in recovery.

The treatment of craniocerebral injuries may be divided into non-surgical and surgical. By far the larger number of cases fall into the former group because there are only three indications for surgery in the treatment of skull fractures These are (1) middle meningeal hemorrhage; (2) subdural and subcortical hematoma, and (3) depressed fractures.

NON-SURGICAL THERAPY

The first consideration in the treatment of a patient who has received a skull injury is to combat even the slightest degree of

shock which may be present as the result of hemorrhage or from the severity of the trauma. Warmth is the most effective single method in these cases unless, of course, bleeding from the scalp or from some other injury has been so severe that it is obvious that a transfusion is required. The recovery from shock, without hemorrhage, which follows a skull injury is usually quite prompt unless the injury is so extensive that death occurs within a short time. This statement indicates that there is a group of skull injuries in which active well-directed treatment is of no avail and death occurs usually within twenty-four hours after the injury.

When blood or cerebrospinal fluid are escaping from the ears or nose, those cavities should be left alone except for a loosely introduced piece of sterile cotton to absorb the discharge. No irrigating solutions of any kind should be introduced into the aural or nasal cavities because of the danger of infection. Examinations of these cavities should be restricted to the absolute minimum, if practiced at all. The adherence to these strict rules is an excellent prophylaxis against a complicating meningitis.

The patient should be placed in a warm bed with the head flat. The use of an ice-bag upon the top of the head is of no value, but the use of an ice-glove over a swollen, edematous cheek or eye aids materially in its reduction.

It is not necessary to rush the patient to the roentgen-ray room for films of the skull. Though a depressed fracture is palpated, it is more important to get the patient into a warm bed than it is to subject him to the manipulations necessary to obtain skull films. Moreover, satisfactory films can be obtained by a portable apparatus which may be taken to the bedside. Even under such circumstances the patient's head should not be forced into unusual positions or roughly handled. Often the slightest movement of a patient's head is followed by a respiratory arrhythmia and collapse of his circulation. When the patient has recovered consciousness and is on the mend, carefully taken lateral, antero-posterior and postero-anterior films may be made. The latter often disclose fractures which cannot be visualized in the lateral position.

The second step to be taken is to reduce edema, brain volume, and intracranial pressure There are two methods employed commonly to bring about these results: (1) the administration of hypertonic solutions and (2) spinal punctures

Hypertonic Solutions.—Weed and McKibben[1] reported that cerebrospinal fluid pressure could be markedly altered by the

[1] Weed, L. H., and McKibben, P. S.: Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injections of Solutions of Various Concentrations, Am. Jour. Physiol., 48, 512, 1919, Experimental Alterations of Brain Bulk, Ibid., p 531

intravenous injections of solutions of various concentrations They showed that the intravenous injection of strongly hypertonic solutions lowered the cerebrospinal fluid pressure to such a degree that often negative readings were recorded. With hypotonic solutions (distilled water) a prolonged rise in fluid pressure occurred. Accompanying these changes in the fluid pressure, Weed and McKibben found marked alterations in the volume of the brain so that the hypertonic solutions produced a small shrunken brain.

These findings have been confirmed many times in the laboratory so that clinical applications of these phenomena have been developed. Cushing and Foley[1] showed that the ingestion of hypertonic solutions reduced cerebrospinal fluid pressure, and Foley and Putnam[2] administered hypertonic solutions intra-intestinally with similar results. Many neurological surgeons have observed the striking decrease in brain volume at the operating table after the administration of intravenous hypertonic solutions in patients with intracranial tumors. Weed and Hughson[3] extended the original observations somewhat in addition to confirming the effects of hypertonic solutions and showed that the cerebrospinal fluid pressure became profoundly lowered, while the sagittal and brachial venous pressures remained about the same. It is, therefore, possible to reduce cerebrospinal fluid pressure without greatly affecting the systemic blood-pressure. These changes in the cerebrospinal fluid, effected by hypertonic solutions, have their explanation in the alteration of the osmotic pressure of the blood The increase in the pressure of the cerebrospinal fluid and in the brain volume may be taken to mean a passage of fluid from blood-vessel to tissue The fall of cerebrospinal fluid pressure and brain volume after the injection of hypertonic solutions points to the attraction of water from the body tissues and possibly from the body fluids.

It was natural that these physiological findings be developed for clinical use It was found early that hypertonic saline solution was dangerous because of its effect upon the kidneys, among other organs. Fifty per cent glucose solution was substituted for intravenous injection. For adults in coma, 100 cc. of 50 per cent solution should be given intravenously every six to twelve hours until the patient becomes coöperative and alert. It may then be given as the patient's condition warrants. It should be given slowly and as

[1] Cushing, H., and Foley, F. E. Alterations of Intracranial Tension by Salt Solutions in the Alimentary Canal, Proc. Soc. Exper. Biol. and Med., 17, 217, 1919

[2] [illegible] The Effect of Salt Ingestion on Cerebrospinal [illegible] Jour. Physiol., 53, 464, 1920

[3] [illegible] Systemic Effects of the Intravenous Injection of Solutions of Various Concentrations With Special Reference to the Cerebrospinal Fluid, Am Jour. Physiol., 53, 53, 85, 130, 1921.

long as thirty to forty minutes should elapse for the injection of 100 cc. For children the dosage should not be over 50 cc. of the same solution, and this may be reduced according to the age of the patient. Clinical experience indicates that the same results cannot be obtained by giving double the amount of 25 per cent solution instead of 100 cc. of 50 per cent solution.

Experimental work by Massermann[1] has shown that there is an initial small rise in the cerebrospinal fluid pressure immediately after the injection of glucose solution which is followed by a fall and a subsequent rise in pressure which may exceed the original pressure. The latter occurs within from two to three hours after the injection of glucose. However, he discovered that by substituting 50 per cent sucrose solution for glucose, the fall in pressure which resulted was greater and was not followed by a rise in pressure which exceeded the original pressure. We can confirm these results clinically and have made use of intravenous injections of 200 cc. of 50 per cent sucrose solution without any apparent untoward effect.[2] After such an injection the fluid pressure begins to rise slowly after about two hours, and consequently if it is desirable to keep the pressure constantly decreased, smaller amounts of the solution may be given at regular three- or four-hour intervals. In connection with these same experiments, Massermann showed that the removal of 20 cc. of spinal fluid by a lumbar puncture in the presence of a normal cerebrospinal fluid pressure would result in a definite decrease in the pressure, which lasted only one hour, but removal of 35 cc. of fluid under the same circumstances was followed by a fall in pressure out of proportion to the added amount removed and is, therefore, dangerous.

Magnesium sulphate (25 per cent solution, 150 to 200 cc.) given by rectum is an effective means of combating increased cerebrospinal fluid pressure and brain volume, but it is expensive in linen and labor and its general use is not particularly satisfactory.

Supportive treatment is important. Fluids should be given to meet the basic metabolic requirements in all patients with cranio-

[1] Massermann, J. H.: Cerebrospinal Hydrodynamics; Effects of Intravenous Injection of Hypertonic Solutions of Dextrose, Arch. Neurol. and Psychiat., 35, 96, 1936.

[2] It has been shown experimentally that the prolonged repeated administration of 50 per cent sucrose solution to dogs produces evidence of permanent glomerular changes in the kidneys Fifty per cent glucose or 50 per cent d-sorbital solutions do not cause these changes It should also be understood that the molecular weights of these hypertonic solutions differ, and, therefore, the volume of the intravenous dosage varies For example, 100 cc. of 50 per cent glucose solution is equivalent in effect to 200 cc. of 50 per cent sucrose solution.

Lindberg H. A., Wald, M H , and Barker, M. H.: Renal Changes Following Administration of Hypertonic Solutions, Arch Int Med , 63, 907, 1939.

cerebral injuries. One thousand cubic centimeters of fluid per day may be regarded as the absolute minimum for an adult and 1500 cc. per day as the desirable average. During hot weather or periods of hyperpyrexia the amounts should be increased to 2500 or 3000 cc. Fluids should never be put into the mouth of unconscious or stuporous patients, but should be introduced by rectal instillations, hypodermoclysis, or very slowly into a vein. Not more than 1000 cc. of the fluids given intravenously in twenty-four hours should be *in the form of physiological saline solution.*

Nutrition must be sustained during the first forty-eight hours after the injury when the patient's absolute metabolism requirements *consist only of fluids and glucose. After that period the* patient will need, in addition, 50 to 60 gm. of protein and sufficient fats to make up his caloric requirements. The fat requirement can be obtained in whole or in part from the fats of the patient's own body, but the proteins must be supplied to the patient in the form of egg albumin by stomach tube, or intravenously in the form of blood plasma or protein preparations. Feedings must be given by tube to the unconscious or stuporous patient because attempts to feed him orally are frequently followed by aspiration and pneumonia. Frequent blood chemistry determinations with specific reference to the blood chlorides, proteins, carbon dioxide combining power and oxygen content are of practical help in maintaining such a patient in chemical balance.

Lumbar Puncture.—A lumbar spinal puncture may be a valuable aid in the treatment of skull injuries, or at the same time it may be most dangerous.

Blood in the subarachnoid spaces is a source of meningeal irritation, the prominent symptoms of which are restlessness and rigidity of the neck. Both of these symptoms vary with the amount of blood present. Extensive basilar injuries with a large amount of blood in the basilar arachnoid cisterns may produce a clinical condition which simulates experimental decerebrate rigidity.

Unfortunately, it has become a common practice to perform lumbar punctures as a *matter of routine to determine the presence or absence* of blood in the cerebrospinal fluid. Careful examination of the patient, particularly if there is an escape of blood and fluid from the ears, makes a lumbar puncture for diagnostic purposes unnecessary.

It is in these patients with blood in the subarachnoid spaces that lumbar puncture has its greatest usefulness as a therapeutic measure. An accurate manometric reading should be made of the initial pressure. Removal of the fluid should be done under constant manometric control so that the cerebrospinal fluid pressure may not be reduced far beyond the normal, which may vary between 70 and 180 mm. of

water pressure. The removal of bloody fluid from a patient who is restless and difficult to control produces striking results. This should be repeated at intervals of eight to twelve hours or less, depending upon the patient's symptoms. It will be found that the fluid becomes less bloody, then xanthochromic, and finally clear as the symptoms disappear.

Lumbar punctures are often employed repeatedly to maintain reduced cerebrospinal fluid pressure. While this may be accomplished for a short period, the pressure does not remain lowered. However, it is possible that lumbar punctures may free the absorptive channels for the cerebrospinal fluid which may be blocked mechanically by the amount of blood present and the reactive cells produced by irritation of the meninges. It must be remembered, however, that in addition to an increase of pressure there exists an increase in brain volume, and it has been proven that hypertonic solutions reduce both brain bulk and cerebrospinal fluid pressure.

Finally, lumbar punctures done without manometric control and in which there is a sudden reduction of pressure at the lowest point in the spinal fluid system are dangerous. Minute hemorrhages in the brain stem and sudden collapse of the cerebellar tonsils into the foramen magnum may occur with a fatal termination. Especially are these circumstances likely to occur in children. The physiological reduction of pressure by hypertonic solutions is certainly more efficient and less dangerous than the mechanical reduction by spinal puncture.

Nursing Care and Drugs.—Patients with craniocerebral injuries must be nursed carefully. Many times the patient is restless only because of a distended urinary bladder or because of bed clothing which has been wet by an involuntary urination. There is perhaps no other one group of patients who generally receive so little careful, detailed nursing attention, particularly in large charitable institutions. Good nursing, with the judicious use of restraints, will make unnecessary the commonly employed narcotic drugs.

Morphine should be absolutely contraindicated in every case of cerebral injury. In the first place, morphine is a medullary depressant and adds only to the depression of the respiratory and vasomotor centers of the medulla. It is common to see a Cheyne-Stokes respiratory rhythm disappear when morphine has been stopped. Secondly, morphine masks the most valuable symptoms of gradually increasing intracranial compression. It is impossible to judge whether the stupor is increasing or decreasing. The contracted pupils which result mask completely the important pupillary dilatation of a localized hemorrhage. Its only advantage is that it produces quiet, which can be effected in the majority of instances by less dangerous

methods. If it is believed that some medication is absolutely necessary, then drugs with less depressant effects upon the medulla should be employed, such as the barbiturate derivatives. However, the use of hypertonic solutions, the removal of blood from the meningeal spaces, and good nursing accomplish this result with far more benefits to the patient, but these methods do require detailed care and attention. Kennedy and Wortis[1] have recommended the administration of caffeine sodiobenzoate in 0.5 gm. doses every four hours for the reduction of intracranial pressure and have presented good evidence in support of their views.

The prevention of aspiration pneumonia in the unconscious patient depends largely upon his position in bed. He should be placed on a firm, flat bed and supplied with a hard pillow. He should never lie on his back, but rather on his side with the upper leg flexed, the body rotated well forward, the face down, and the jaw and tongue dependent. In this position all mucus from the uppermost lung and bronchi, the trachea, pharynx, and the mouth will drain freely out and the tongue will not interfere with the airway. The patient should be turned from one side to the other every hour at least, day and night.

Frequent turning also aids in the prevention of pressure sores of the skin and subcutaneous tissue In each position, bony prominences must be protected from direct pressure of the bed or from other extremities. Tissues which have born the weight of the body should be massaged gently with a well-greased hand after each turning to restore the circulation more fully.

SURGICAL TREATMENT

The recognition of a craniocerebral injury presents no problem but the evaluation of the extent of the injury is sometimes difficult or impossible during the first few hours after the injury. Intracranial hemorrhage is potentially present in every patient who has received a blow to the head regardless of the presence of recognizable damage to the scalp or skull. *Small lacerations of the scalp may be* associated with extensive fissure fractures, comminuted, or depressed fractures; or, with serious intracranial bleeding. This is particularly true of penetrating wounds inflicted by high velocity small missiles.

The basic principle in the treatment of craniocerebral injuries is the removal of the patient at the earliest possible moment to a hospital where definitive intracranial surgery can be performed to

[1] Kennedy, F., and Wortis, S. B. How to Treat Head Injuries and Appraise Them, Jour Am Med Assn., 98, 1352, 1932.

the extent that it is needed. Emergency treatment should have for its sole purpose the preparation of the injured man for this evacuation. It has been proven that a patient with a craniocerebral injury stands transportation well before a definitive operation has been performed.

Immediate Treatment

The hair should be cut over a wide area about the wound with clippers or scissors and shaved with a razor as carefully and closely as time and the situation permits. The wound should be protected during this procedure with a sterile gauze dressing. This area of scalp should then be washed quickly but meticulously with soap and water. Bleeding from wounds of the scalp can usually be controlled by firm pressure gauze dressings. Ligature of the larger arteries of the scalp is unsatisfactory and often impossible because these vessels retract into the scalp. The wound should not be explored, irrigated, debrided, or sutured until the patient reaches the point chosen for definitive surgical treatment. An ample dressing of gauze should be large enough to cover the wound completely and should be firmly applied with a bandage, not adhesive tape, which covers the entire head. If the patient must be moved a considerable distance, this dressing can be reinforced with an elastoplast dressing to keep it in place without a change until the patient reaches the hospital where definitive treatment is to be given.

A considerable amount of blood may be lost by hemorrhage from the scalp and it should be replaced exactly as it is in wounds of other parts of the body. Bleeding from the ears should be treated by covering the external ear with a large loose gauze dressing and no effort should be made to stop the bleeding. The auditory canal should not be explored, mechanically cleansed, or irrigated.

Shock is not commonly the result of the craniocerebral injury. Its presence usually indicates bleeding or trauma in other parts of the body. In such cases, however, the head should not be placed lower than the trunk and extremities because this position tends to aggravate intracranial bleeding. Otherwise, shock associated with craniocerebral injuries should be treated without regard to the brain injury Blood and plasma should be given as they are needed until the blood pressure level approximates normal.

The unconscious patient should not be transported lying upon his back. In that position he may aspirate mucus and vomitus into his lungs and may have his airway obstructed by the tongue. He should be horizontal, lying on his side, his body rotated well forward, his upper thigh flexed on the pelvis to hold him, his head

resting on a firm pillow or folded blanket and turned toward the ground. This position prevents the tongue from falling back and interfering with respiratory exchange. It will also prevent aspiration of mucus and vomitus and will greatly reduce the hazard of aspiration pneumonia. In compound wounds the patient should lie only upon the uninjured side of the head unless a complicating thoracic wound contraindicates that position. Conscious patients may have the head and shoulders elevated.

Morphine and other opium derivatives are contraindicated in cases of severe craniocerebral injury because of their strong depressant action on the respiratory center of the brain. The soporifics, such as chloral hydrate, paraldehyde and amytal are contraindicated because their use masks the significant changes in the patient's state of consciousness which are of prime importance in determining the presence of intracranial bleeding and the indications for operation. Fortunately, pain is rarely a serious problem in craniocerebral injuries unless they are complicated by serious injury to other parts of the body. When wounds of the thorax, abdomen, or extremities do cause great pain the first consideration should always be to relieve the pain but to use the smallest, effective amount of sedative.

*Often restlessness presents a serious problem in the care of cranio*cerebral injuries particularly during transportation. Sodium luminal, 3 to 5 gr. (0.2 to 0.3 gm.) given intravenously is the best drug to allay restlessness in that it will quiet the patient without depressing the respiratory center or greatly disturbing his state of consciousness. Sodium bromide, or the triple bromides 15 to 30 gr. (1 to 2 gm.) by mouth or 45 to 60 gr. (3 to 4 gm.) by rectum are also of value.

One thousand to 1500 cc. of fluid each twenty-four hours should be given *to all patients and, if they are unconscious or vomiting,* this should be given by rectal drip or by slow intravenous administration. High caloric feedings should be given by stomach tube, if necessary.

The first doctor to treat the patient with a serious craniocerebral injury should record the following facts without fail: (1) the time of injury as nearly as can be determined; (2) the state of consciousness; is the patient rational, restless, confused, drowsy, stuporous or comatose? (3) paralysis; does the patient move both arms and both legs voluntarily or in response to painful stimuli? (4) aphasia; does the patient talk or is he conscious but unable to talk? (5) pulse rate counted for half a minute; (6) respiratory rate counted for a full minute; (7) blood-pressure.

Transportation to a hospital where definitive neurosurgical care can be given the patient should be initiated as early as possible, and

air transport below 5000 feet altitude is perfectly safe. These patients tolerate transporation very well, particularly before a definitive operation has been performed. It is more important that definitive care be given by a well-qualified and experienced neurological surgeon under favorable conditions than that it be given early. With antibiotic therapy a primary closure of craniocerebral wounds can be carried out safely as late as forty-eight to seventy-two hours after injury.

Definitive Treatment

Scalp Wounds.—Every wound of the scalp, no matter how small, may be a penetrating wound with injury to the brain. If the nature of the injury and careful inspection of the exposed skull do not suffice to make a decision possible, roentgen-ray films of the skull should be taken before definitive treatment is begun to establish the presence or absence of indriven fragments of bones or missiles.

Primary closure of the scalp wound should be attempted in every case even as late as twenty-four hours following the injury in wounds which have not received adequate immediate treatment, provided vigorous systemic antibiotic and chemotherapy is instituted after the operation. Wounds which have received preliminary treatment and antibiotics and chemotherapy systemically may be closed forty-eight hours or later after injury. A wide area of hair should be clipped away, and the scalp should be shaved closely and carefully. This area should then be cleansed carefully with soap and water until it is surgically clean. After conservatively debriding the edges of the wound, interrupted silk sutures should be introduced to close the galea and finally, the skin edges. The latter sutures may be removed within forty-eight hours to prevent cross scars, and the edges of the healing wound can be supported with narrow collodion gauze strips to prevent separation. Drainage should be employed but rarely with wounds of the scalp and only for the express purpose of relieving tension by evacuating fresh blood. Drains do not prevent infection, they frequently introduce it. When they are used in non-infected wounds of the scalp they should be led out through stab wounds and removed not later than twenty-four hours after the operation. If avulsion of an area of the scalp has occurred, a firm pressure dressing using sterile mechanics cotton waste should be applied. The introduction of silkworm gut or catgut sutures from the skin through the galea and out again is evidence of a gross surgical technique which should be avoided. Antibiotic and chemotherapy is indicated in all cases of badly traumatized and contaminated wounds of the scalp and cranial vault.

Linear Skull Fractures.—Simple linear fractures of the skull are of little consequence in themselves, apart from the concomitant injury to the intracranial contents. However, they are frequently associated with intracranial hemorrhage; those of the vault with epidural bleeding from a torn middle meningeal artery and those at the base with bleeding into the basilar cisternæ from veins leading into the cavernous and sigmoid sinuses.

Compound linear fractures are important because they afford a portal of entry for infections into the intracranial cavity. This is particularly true of fractures compounded through the cribriform plate, the accessory air sinuses, or through the external auditory canal. It is important therefore to give adequate antibiotic and chemotherapy in all cases of compounded linear fractures. *Bleeding from the ear is an indication of a compound fracture into the external auditory canal*

Depressed Skull Fractures.—Depression of one or both tables of the skull may occur without a scalp injury. It is sometimes difficult to determine the presence of a depression without carefully made roentgen-ray films of the skull. Often upon palpation, the periphery of an extensive hematoma of the scalp will give almost the exact impression of a large depression of the bone. This is particularly true in infants and children, and therefore, roentgenograms should be used to check the diagnosis.

It is the simple depressed fractures which require careful and conservative judgment concerning their treatment. *Certainly not every* depression of the skull should be operated upon. Rather pronounced depressions of the bone may not produce the slightest neurological symptom To operate upon each patient because of the anticipation of epilepsy, or other evidences of a cortical lesion, is to disregard the relative small percentage of relationship between these symptoms and extensive skull injuries. If obvious symptoms are present with a depressed fracture, the defect should be remedied. A small scalp-flap incision should be made with the depression as the center. Persistent attempts to pry the fragments back into position should not be made There is no reason why the fragments should not be approached by a rongeur through a burr-hole opening in the adjacent normal skull and removed. Less damage to the underlying dura and brain are likely to follow the latter procedure.

Large extensive wounds of the scalp and skull may be accompanied by comminution of the bone fragments, some of which may be driven through the dura mater into the brain along with other foreign materials These ragged, lacerated wounds of the scalp should be cleaned with soap and water, irrigated lightly with saline solution, and the surrounding scalp should be shaved carefully.

Pieces of indriven bone, hair, metallic fragments or clothing should be removed with a fine forceps. A soft rubber catheter may be passed through the opening in the dura mater and into the wound in the brain. By irrigating gently through the catheter with sterile saline solution and by gentle suction with a glass syringe and bulb, or a not too powerful suction apparatus, pulped brain and small pieces of bone will escape from the wound in the brain.

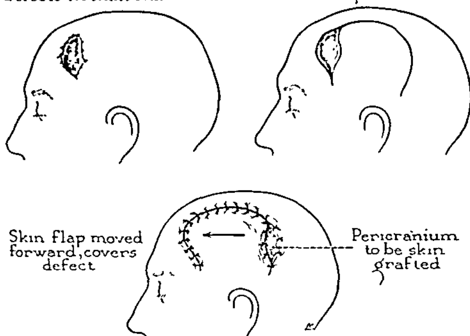


FIG. 42.—Method of closing defect in scalp by "sliding flap" method. (Courtesy of Dr. John E. Scarff and Surg., Gynec and Obst.)

Incisions should then be planned to close the wound edges without tension. A simple vertical or curvilinear incision is most satisfactory to insure healing. Quite often so much scalp is destroyed that direct closure, after debridement, cannot be carried out. The "tripod" incision, or its Isle of Man modification; one or more relaxation incisions, S-shaped incisions, or a large flap may be used. Great care must be used in handling the scalp flaps to avoid bruising their apices. The flaps should be sufficiently long for the elasticity of the scalp to allow stretching over the defect without tension.

Large defects in the dura mater should be closed by "split-dural" flaps, or with free transplants from the temporal fascia or the fascia lata

Often the defect in the scalp may be so large that the tripod incision method of closure is impractical. Under these circumstances

a "sliding flap" of scalp which has the shape of an elongated horseshoe (Fig. 42) and includes the pericranium may be used. In outlining and cutting such a flap it is important to make it considerably oversized in both transverse and longitudinal directions, because of the complete inelasticity of the tissue. If great care is not exercised in doing this, the edges intended to be apposed will not meet. The base of the flap should be wide to insure an adequate blood supply to its periphery. Edges of the flap not sutured to the scalp should be loosely sutured to the pericranium. Drains need not, and should not, be used. The pericranial surface left exposed after moving the flap should be covered immediately by a Thiersch graft, or a skin graft of intermediate thickness. Such grafts are successful in a high percentage of cases. In rare cases in which they do not succeed the exposed bone will be covered quickly with granulations if numerous small drill holes are made through the outer table to the diploic spaces. These granulations can subsequently be covered with pinch grafts or other types of skin grafts.

Compound comminuted fractures into the accessory nasal sinuses present troublesome problems. No rule can be laid down for treatment which will meet the specific needs of every case. In general, however, the principle should be to repair the scalp and dura mater exactly as in compound, comminuted fractures in other parts of the cranial vault and to do as little as possible to the sinus. In dealing with the sinus it is *axiomatic that the less surgery, the less trouble.* The contour of the sinus should be re-established as far as possible by gentle manipulation with the smooth rounded end of a hemostat or other similar instrument. Comminuted portions of the bony walls should be preserved wherever possible and only fragments of bone entirely detached from all soft tissues should be discarded. The mucous membrane lining the sinus should be carefully conserved, even though it is badly torn, because of the great power of regeneration possessed by this membrane and its strong tendency to reform a functioning air sinus. The frontonasal ducts should not, as a rule, be touched. Drains should not be used. The danger of serious intracranial infection is generally less than might be reasonably expected if systemic antibiotic and chemotherapy is aggressively employed Plastic repair of defects remaining in the frontal bone after compound comminuted fractures in this region should not be attempted at the time of the acute injury, but should be delayed for several months until all danger of local sepsis is passed. At that time repair may be made by any one of several methods to be discussed later

Rhinorrhea of cerebrospinal fluid persisting for longer than ten days should be treated by intracranial closure of the internal orifice

of the fistula by a fascia transplant as soon as a craniotomy may be performed without undue danger of infection. Maximum chemotherapy and antibiotics should be started early and maintained for a long time.

Penetrating Wounds of the Skull.—The basic principles outlined for the treatment of wounds of the scalp, compound fractures of the skull, cerebral edema, and intracranial hemorrhage apply equally well to the treatment of penetrating wounds of the skull. Roentgenograms of the skull should be taken before definitive treatment is undertaken.

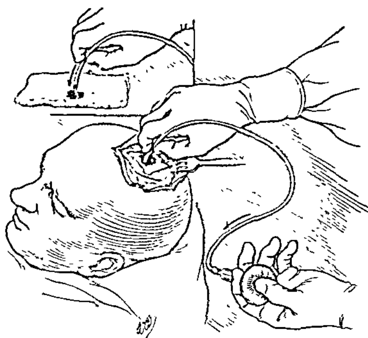


Fig. 43.—Method of suction of the tract of a penetrating wound in the brain. (After Cushing.)

For the purpose of following a specific line of treatment, penetrating wounds received shortly after injury may be distinguished from those which are treated more than seventy-two hours following injury. The early cases include those which have not received preliminary immediate treatment with chemotherapy and antibiotics but which present themselves for definitive treatment not later than thirty-six hours after the wound was received and without signs of active sepsis in the wound at that time and those cases which have received adequate immediate chemotherapy and antibiotics therapy at the time of injury, or soon after, and present themselves for definitive treatment not later than seventy-two hours after the injury and without signs of active sepsis in the wound at that time.

7

The entire head should be shaved immediately prior to operation. Dirt and foreign matter external to the dura mater should be removed and the wound thoroughly cleansed with physiological saline solution. Debridement of the skin should be conservative. A large area of the scalp should be cleansed and so draped that any plastic procedure may be performed which might be necessary to close the wound, such as a large sliding scalp flap. All loose fragments of bone should be discarded. The openings in the skull and dura mater should be enlarged sufficiently to allow exploration of the tract of the missile within the brain. Devitalized brain tissue should be excised. The tracts of all large missiles within the brain should be explored insofar as this can be done easily. Dirt and fragments of missiles, bone, and other foreign materials should be picked out and the tract thoroughly irrigated with physiological saline solution. Damaged and contaminated cortical tissue lining these tracts should be removed with the suction apparatus. (Fig. 43.) The opening in the dura mater should be closed tightly by the use of free grafts of the patient's temporal fascia, or of fascia lata, held in place with fine silk sutures. Primary suture of the scalp must be accomplished over defects in bone and dura mater.

The removal of bony fragments from the brain is of the utmost importance since experience has shown that cerebral abscesses develop around 50 per cent of bone fragments not removed. The smaller metallic fragments constitute no such danger and attempts to remove them should not be made at the risk of damaging healthy cerebral tissue. Cerebral abscess should be suspected whenever normal recovery from a penetrating wound of the head is arrested and if necessary for diagnosis, pneumoventriculography may properly be performed

Late penetrating wounds of the head are those not included in the above description and the majority will be septic. In addition to cellulitis of the scalp many will already have developed meningitis, cerebritis, cerebral abscess, osteomyelitis, cerebral herniations and fungi Osteomyelitis of the skull requires free dependent drainage and removal of sequestra as they form, in addition to intensive chemotherapy and antibiotics. Meningitis and cerebritis also should be treated vigorously with chemotherapy and the antibiotics and respond favorably in a majority of cases In the case of early abscesses secondary to penetrating wounds, exploration of the tract with removal of bone fragments and foreign material, followed by a short period of drainage, is the method of choice

Pathology and Treatment of Cerebral Fungus.[1]—Cerebral herniation and fungus formation may occur whenever there is a defect

[1] Martin, J. and Campbell, E. H *Early Complications Following Penetrating Wounds of the Skull*, Jour. Neurosurgery, 3, 58, 1946.

in the dura mater, bone and scalp overlying the brain and there is a rise in intracranial pressure. Cerebral herniations invariably become infected and there is usually some necrosis of superficial tissue due to interference with the blood supply. A cerebral herniation which becomes infected and partially necrotic is termed a cerebral fungus.

Cerebral fungi may be of two kinds. The first consists of a localized cerebritis which follows a superficial scalp infection and a torn dura. The herniation is usually not large or friable. High intracranial pressure or neurological signs may not be present. When there is an increase in intracranial pressure, there is usually an associated deep infection, hematoma or necrotic brain tissue and the fungus develops rapidly, is shaggy and bleeds easily. Retained metallic and bone fragments should be sought for. Often a sinus may be discovered which leads from the fungus to a deep abscess.

If the intracranial pressure returns to normal, the process of extrusion of the cerebral cortex gradually ceases, the necrotic tissue is removed by natural processes and the surface of the brain becomes covered with granulation tissue and eventually, scar tissue. This eventually contracts with sufficient force to counteract the expanding action of the intracranial pressure and will, in favorable cases, finally reduce the hernia completely to the general contour of the skull. It remains only for the surface of the scar tissue to become epithelialized and this may take place spontaneously around the edges of normal skin, or may have to be accomplished by grafting.

The primary object in the treatment of cerebral herniation is to keep to a minimum the protrusion of cortical tissue through the skull defect. In attempting to accomplish this, efforts should be directed primarily at minimizing expanding pressure within the skull, rather than attempting to hold the brain within the cranial cavity by applying pressure against it from without by means of pressure dressings. This latter procedure is almost invariably ineffective against continuous, elevated intracranial pressure and in addition, causes pressure necrosis of the herniating brain and greatly increases the amount of local sepsis in the brain tissue.

The patient should be kept in a sitting posture in bed whenever it is possible as a method of aiding the reduction in intracranial pressure. Thirty to 50 cc. of spinal fluid should be removed once or twice daily for three to four weeks until the necrotic and infected tissue has been removed from the surface of the herniated mass and the exposed brain is covered with a clean layer of granulation tissue. Coughing, vomiting and straining at stool must be prevented. The patient should be guarded against acquiring an upper respiratory tract infection which will force him to cough and violently force a protrusion of the hernia.

Wet compresses, Dakin's solution or sulfonamide dressings should be applied to the surface of the herniation or fungus until the exposed brain is covered with healthy granulation tissue. Thereafter, lubricated dressings which will not interfere with the ingrowth of epithelium from the margins of the wound and can be changed without injuring the new granulation tissue should be used. Superficial marginal abscesses often occur beneath the overhanging edges of the scalp, bone and dura mater and must be drained effectively but they can be prevented by having the bone opening larger than that in the dura mater and the scalp opening larger than the bony opening. A firm ring of cotton or gauze about the herniation will protect it from direct pressure which will produce necrosis and favor the development of a local infection. The loss of superficial tissue by necrosis and infection brings the lateral wall of the ventricle closer to the surface and with it rupture of the ventricle and leakage of cerebrospinal fluid makes the danger of meningitis more imminent.

A persistently progressive herniation following three or four weeks of proper treatment or an excessively rapid protrusion is an indication of an elevation of intracranial pressure which is usually due to an extradural, subdural or intracerebral abscess. Roentgen-ray evidence of retained bone fragments usually points to the site of the abscess which must then be drained adequately.

Intracranial Hemorrhage.—Intracranial hemorrhage must be suspected in every patient who becomes progressively worse following a blow to the head and it may be epidural, subdural, intracortical or intracisternal in its location.

Epidural hemorrhage should be suspected with fractures of the vault of the skull and the bleeding is arterial from the torn branches of the *middle meningeal artery*. The three most characteristic symptoms of this condition are: (1) dilatation of the pupil upon the side of the *hemorrhage;* (2) gradual onset of coma with or without an early lucid interval; and (3) the presence of signs of motor weakness on that side of the body opposite to the dilated pupil. The location of the external injury to the scalp, or of the skull fracture, must not influence the localizing diagnosis in these cases.

In a typical case, the diagnosis of a middle meningeal hemorrhage is not difficult and the usual period of consciousness which immediately precedes the progressive development of stupor is pathognomonic. Unlike patients with lacerations of the brain, the temperature usually is only slightly elevated. The patient is drowsy but can be aroused, and the appearance is apt to mislead one into believing that he is in good condition. The significance of a dilated pupil upon the side of the hemorrhage is not sufficiently appreciated It is not difficult to understand how a hematoma, dissecting its way

between dura mater and bone, extends forward and medially into the middle fossa. Reaching the superior orbital fissure, pressure upon the oculomotor nerve is produced, and it is surprising that the trochlear and abducens nerves are not involved more often with the oculomotor. The size of the latter may afford protection to the trochlear to a great extent, and because of its lateral position the abducens may be protected by the reflected edge of the dura. The base of the middle fossa of the skull is very vascular, and as the dura mater is dissected away by the clot, bleeding from the vascular channels in the bone adds to the rapidly advancing hematoma.

Because of the oculomotor palsy, the eyeball may be drawn downward and laterally by the unopposed action of the superior oblique and external rectus muscles, innervated by the trochlear and abducens nerves. Ptosis of the upper eyelid may escape notice unless a careful examination is made for the presence of the slightest neurological signs.

If the clot extends upward over the convexity of the cerebral hemisphere or if the brain is pushed upward and medially by the hematoma, symptoms of a hemiparesis may gradually develop on the opposite side of the body. The face is usually involved first, and in turn the arm and leg. Oculomotor nerve paralysis plus a contralateral hemiparesis, or paralysis, constitutes a syndrome described by Weber and is usually attributed to a lesion of the midbrain at the level of the oculomotor nerve nuclei. The duplication of this syndrome by a middle meningeal hemorrhage is an important diagnostic fact to remember from the standpoint of surgical therapy.

The insidiousness of the onset of the symptoms of a middle meningeal hemorrhage are well illustrated by this young patient's story:

At noon on November 20, 1938, a twelve-year-old girl fell from a horse and received several abrasions of the skin on her forehead and about her mouth and chin but did not lose consciousness. She remounted her horse, rode back to the stables and then went home. At 4 o'clock that afternoon she complained of headache, and at 6 o'clock her mother had difficulty in arousing her. Throughout the evening she was drowsy, restless, and complained of headaches At 3 o'clock in the morning she was deeply unconscious and had an epileptiform seizure. It was not until 5 o'clock on November 21 that it was recognized that her right-sided Jacksonian convulsive seizures, paresis of the right side of the face and of the right arm and leg, dilatation of the left pupil, absence of the right abdominal reflexes and a right Babinski reflex were evidences of a middle meningeal artery hemorrhage, which was found at operation to have dissected into the middle fossa of the cranial cavity.

Middle meningeal arteries may vary enormously in their relations between the two sides of the head. The most common type is that which has an anterior and posterior branch which arises from the main trunk as it lies in the middle fossa, near the foramen spinosum.

or farther up on the dura mater covering the lateral surface of the temporal lobe. However, there may be two or more branches running anteriorly and posteriorly. The fact that the parent trunk or any of these branches may be torn should indicate definitely the character of the operation which should be performed.[1]

A typical osteoplastic craniotomy operation should be done with the temporal bone as the center of the bone flap. Attention should be paid to all of the meticulous details of such a procedure. There should be no bleeding from the scalp during the operation if proper hemostasis is employed. With the flap elevated the clot should be irrigated away with normal saline solution and the origin of the bleeding discovered. If it is on the surface of the dura, a fine cambric silk suture or a silver clip may be used for ligation, or the vessel may be coagulated with an electrosurgical unit. It may be necessary to elevate the dura from the floor of the middle fossa and compress the vessel into the foramen spinosum by bone wax in order to control the bleeding successfully. If very small oozing points still remain on the surface of the dura, gelfoam stamps prove very efficient in controlling the bleeding The bone and scalp flap should be replaced and sutured with interrupted cambric silk sutures, layer by layer. Hemostasis must be perfect, and drainage material should not be introduced into the wound. Any operative procedure which does not afford a rather extensive view of the middle cranial fossa and the convex surface of the cerebral hemisphere is likely to be accompanied by serious difficulties.

A rather trivial injury to the head may be followed, in certain individuals, by symptoms of cortical irritation and of increased intracranial pressure due to a *subdural hematoma*. Aside from trauma, there is no single etiological factor in these cases. A fall from a stair step, a slip on the ice with the buttocks as the point of impact, a slap in the face may initiate a hemorrhage. Only one of our patients was an alcoholic although chronic alcoholism has been thought to be a factor.

In the early literature, little or no attention was paid to post-traumatic hematomas. Virchow had so accurately described what he termed pachymeningitis hæmorrhagica and called the bloody cysts, which were present, hematomas of the dura However, Virchow did not describe a traumatic subdural hemorrhage but theoretically discussed the difference between such a condition and

[1] The common assumption is that extradural hematomas are caused by a rupture of the middle meningeal artery or one of its branches, but it must be remembered that in some instances the source of the bleeding which strips the dura mater from the bone is venous That is, the venous sinuses around the middle meningeal or its branches may be torn and the arteries may be intact

what he spoke of as a spontaneous hematoma. Putnam[1] has pointed out that the post-traumatic hematoma is distinguished from the spontaneous form by histological as well as clinical peculiarities.

In from one-third to one-half of all cases the process is bilateral, and this is of particular surgical importance. Hematomas are more common in the parietal region, but in many cases the middle and frontal fossæ of the skull are packed with a thick hematoma. Usually, the clot is enclosed in a fibrous tissue envelope, which may be slightly adherent to the dura but not to the arachnoid. On the arachnoid surface, the membrane consists of a thin sheet of connective tissue covered by a single layer of mesothelial cells. The most characteristic feature of the membrane on the dural side is the large, irregular spaces which have a mesothelial lining. These spaces are parallel to the surface of the dura and are separated from it by a few fibroblasts. Although they may appear empty, the spaces usually contain red cells with an occasional leucocyte, fibrin, or granular debris. Putnam believes that these spaces communicate one with the other and with the neighboring capillaries. In general, the longer the interval between the trauma and removal of the enveloped hematoma, the larger and more irregular are the spaces. It is in these respects that the membrane of the traumatic hematoma differs from the commonly described pachymeningitis hemorrhagica interna. In the latter, thin-walled vessels are enormous and no mesothelium lined spaces are present.

In some cases in which the onset of symptoms is delayed for only a few days following the trauma, it is possible that the bleeding occurs immediately after the accident, and symptoms develop only when the brain becomes edematous. In the more chronic cases, the hematoma may be very thick, olive-green and mottled, and this rather characteristic color is transmitted through the dura mater. In some instances the subdural hemorrhage is acute in that bleeding is rapid and extensive so that symptoms develop after a comparatively short interval following the exciting trauma. These hematomas consist of dark, partially clotted and liquid blood which is removed quite easily. They must be distinctly separated from the more chronic hematoma in which bleeding is slow and organization takes place to be followed again by fresh oozing.[2]

As Putnam has said, "The mechanism of production of the far more common posttraumatic extradural extravasations is well understood, and the process of organization of the clot is the same as that

[1] Putnam, T. J., and Cushing, H.: Chronic Subdural Hematoma, Arch. Surg., 11, 329, 1925.

[2] Kennedy, F., and Wortis, H: "Acute" Subdural Hematomas and Epidural Hemorrhage, Study of 72 Cases of Hematoma and 17 Cases of Hemorrhage, Surg., Gynec. and Obst., 63, 732, 1936.

of a hematoma elsewhere in the body. That a clot should slowly form in the mesothelium lined subdural space is quite a different matter. If such clots were likely to follow trauma, one would expect to find them more often after fractures of the skull, and they would be a frequent postoperative complication of intracranial osteoplastic explorations. . . . It would appear, therefore, that in the disorder under consideration there must be some individual predisposition of the meninges favorable to the subdural seepage of blood.

"In certain cases in which the onset of symptoms is delayed for only a few days after trauma, it is possible that they may be due to a hematoma formed immediately but which only announces its presence when the brain becomes edematous or congested. In other cases the occurrence of late hemorrhages seems probable and may be due to the formation of communication between the mesothelium lined spaces and blood-vessels, with rupture of one or the other."

The interval between the injury and the onset of symptoms may be days, weeks, months, or even years. Three months elapsed between a fall down stairs and the onset of headaches in one of our patients, who came to the hospital with a hemiplegia, aphasia, and was semi-stuporous. Naturally, the longer the interval, the more gradual is the onset of symptoms. In fact, one of the most striking characteristics of a subdural hematoma is the extreme variability, vagueness, and fluctuability of the symptoms from day to day or hour to hour and this fact, in itself, is a diagnostic point of the greatest value. Any combination of symptoms of general intracranial pressure, or local pressure, may be present in atypical, incomplete or fleeting variations. Often these patients are disoriented, irritable, *and many forms of mental disease may be imitated. The spinal fluid may be entirely clear, or only slightly yellow but usually is under* increased pressure. There is no other intracranial lesion so difficult to diagnose accurately upon the subjective and objective symptoms alone and often a hematoma may be encountered when it is least expected

Our experience in two instances serves to emphasize a difference in the onset of symptoms and a difference in the character of the hematomas, which is an important factor affecting the type of operation which can be carried out successfully.

On December 23, 1937, a fifty-six year old painter fell from his ladder about 6 feet to the floor He did not strike his head but fell on his right shoulder He was able to arise and go on with his work He complained for the next three days of a mild headache, an earache, and a painful shoulder He continued to work, but on January 1 at noontime he appeared dazed and had difficulty in speaking His family stated that he sounded as though his tongue were swollen, and although he appeared to know what he wished to say, it was

difficult to understand him. On the following day he complained of numbness of the right arm and movements of that hand were clumsy and weak. He continued to be up and about his home, but his speech became more impaired and his headaches more severe. On January 4 he became unresponsive, stuporous, and could be aroused for short periods only with difficulty

On January 7 he had developed weakness of the right side of the face, a right hemiparesis, absence of the right abdominal reflexes and was unable to respond, although he could be aroused and endeavored to obey commands. Burr holes were placed in the skull over the frontal and parietal areas and a hematoma was found containing dark partially clotted and fluid blood. The blood was removed by suction and by irrigation between the burr hole openings until it had been removed completely. Recovery was prompt and uneventful.

In contrast another patient developed symptoms over such a long period of time that the original trauma had been forgotten, and his hematoma was thick, yellowish-green in color and had seriously deformed the underlying brain.

For six weeks prior to March 15, 1937, the patient complained of a frontal headache, but he kept at his work as a laborer. On that night he was restless in bed and finally arose. He wife found him some minutes later in the basement unable to use his left arm and leg. It was not until March 23 that upon examination it was found that the left palpebral fissure was larger than the right; the optic disc edges were blurred and slightly elevated; no motion was possible in the left upper extremity which was strongly flexed and adducted; there was marked weakness in the left lower extremity; the left deep tendon reflexes were increased, and the left abdominal reflexes were absent. The patient yawned frequently, and this act was often associated with flexion of the left upper extremity. Often he responded to commands and attempted to answer questions. There were periods of marked spasticity in the left upper extremity which would disappear and it would become flaccid.

It was thought that the patient had an intracranial tumor although the alternating spasticity and flaccidity in the [illegible]
At any rate, an osteoplastic craniotomy w[illegible]
membrane was found beneath the dura. [illegible]
fluid escaped This hematoma covered the entire right cerebral hemisphere and had stained the surface of the cortex a yellowish-green. The brain had a doughy appearance, and it did not regain its normal consistency quickly. The patient had a stormy convalescence but progressively recovered until he was completely well after two weeks.

This case illustrates how difficult it is at times to make a positive preoperative diagnosis of a subdural hematoma, particularly when the interval between a possible etiological trauma and the development of symptoms is long. It has become a general practice, therefore, to place small craniodural openings in the four quadrants of the skull when the presence of a subdural hematoma is suspected, and the condition should be suspected in every patient with a head injury when satisfactory initial progress becomes arrested, or when the patient's state actually deteriorates. A small burr hole opening

through a small incision made under local anesthesia and a small opening in the dura mater will quickly disclose the characteristic color of a recent or chronic hematoma. We have found, with Fleming and Jones[1] that removal of the clot by aspiration and irrigation between the openings with warm saline solution is a very effective method of treating a recent hematoma in which the contents of the limiting membrane has not become organized. Thick, organized hematomas in which the liquid portion represents only a small part of the pathology present, cannot be so satisfactorily handled in this manner. It becomes necessary to remove the clot more completely, and to do so requires an osteoplastic craniotomy. The mottled, yellowish-green hematoma can be peeled off the underlying cortex rather easily and resembles a fresh piece of liver in its consistency. Often in the chronic hematomas we have found the middle and anterior cranial fossæ filled with the thick, organized clot. Hemostasis must be very painstaking, and if the hematoma is removed carefully and completely, there is little danger of its re-formation.

On the other hand, cerebral edema may occur in the brain which is suddenly released from a long continued pressure. One's first thought upon observing the recurrence of coma, or of convulsions after operation, is that bleeding has recurred. Secondary operations and autopsies have shown, however, that this is not the case but that the brain is tense and the convolutions are flattened by edema. It may be advisable to leave a stellate-shaped opening in the dura mater over the temporal lobe and to remove the squamous portion of the temporal bone from the bone flap, thus affording a decompression opening. The additional use of hypertonic solutions intravenously will aid in the treatment of the patient, but extreme caution and good judgment must be used, particularly if after operation the patient's blood-pressure fluctuates through wide ranges in short periods of time.

The brain usually expands to fill the cavity left by removal of the hematoma, but not infrequently it fails to regain its size and remains so completely compressed that it appears as if the convolutions have been hardened in their deformed condition. In our experience, when the brain does not begin to expand into its normal shape rather promptly after the removal of a hematoma, the patient's convalescence is usually very stormy. Two of our patients died following the removal of chronic subdural hematomas and in both instances the deformed character of the convexity of the hemisphere was still

[1] Fleming, H. W. and Jones, O. W.: Chronic Subdural Hematoma, Simple Drainage as a Method of Treatment, Report of Eight Cases. Surg., Gynec. and Obst., 54, 81, 1932.

present three days and ten days following operation and removal of the hematoma.

If it is arterial in origin, *intracortical hemorrhage* is likely to dissect into the ventricles and be rapidly fatal. If venous in origin, it tends to form large subcortical clots which occur in the temporal lobes in about 90 per cent of the cases and equally upon the right and left sides and with almost equal frequency on the side opposite the blow as on the side of the injury. These venous subcortical clots are characterized by a preponderance of focal signs over those of generalized intracranial pressure which is the reverse of the clinical picture produced by epidural or subdural collections of blood. An exploratory puncture or an incision into the temporal lobe will allow them to be evacuated and the prognosis for the recovery of function is excellent.

Intracisternal hemorrhage is frequently associated with basilar skull fractures and bleeding usually occurs from a tear in one of the large communicating veins which leads from the cortex into a dural sinus and takes place directly into one of the subarachnoid cisterns. Blood in the cerebrospinal fluid tends to block the channels of absorption over the cerebral hemispheres and an acute hydrocephalus results which leads to a fatality without the possibility of successful surgical treatment.

Subtemporal Decompression.—It should be noted that a subtemporal decompression has not been described as a method of therapy. No matter how carefully performed and in the hands of a surgeon experienced in handling nerve tissue, some edema will follow this operation This adds only to that intracranial pressure which already exists. It has been proved that the mortality rate following the treatment of skull fractures has been lowered tremendously since the abandonment of the indiscriminate use of decompression operations for the relief of traumatic edema.

Concomitant Injuries.—Many times a craniocerebral injury so dominates the clinical picture that other serious injuries are overlooked. The patient must be examined carefully for fractured ribs, fractures of the bones of the extremities, and injuries to the viscera, particularly the urinary bladder and abdominal organs. We have seen a ruptured spleen in a child who remained in coma for several days following a skull injury. It was quite obvious from her severe shock, rigid abdomen, and marked anemia that an intra-abdominal lesion was present. Fractures of the phalanges of the fingers, metacarpal and carpal bones occur not infrequently. In fact, the number of concomitant injuries is legion, and unless recognized and treated promptly and effectively they, and not the cerebral injury, may decide the issue.

TREATMENT OF COMPLICATIONS

Meningitis.—*The* entire clinical story of the treatment of meningitis has changed following the use of the antibiotics and chemotherapy in the treatment of meningitis and craniocerebral wounds during the last war.

Penetrating wounds of the brain, rupture of the wall of the lateral ventricle following cerebral herniations or fungi, fractures into the paranasal sinuses and middle-ear infections are the most common sources of meningitis following craniocerebral injuries. Infection develops within the substance of the brain in penetrating brain wounds and the result is a brain abscess or a spreading purulent encephalitis which is often associated with fungus formation, purulent ventriculitis, meningitis and death. Meningitis may arise in wounds of the orbit and frontal bone without an intervening brain abscess or ventricular infection.

The sulfonamide drugs, preferably sulfadiazine, should be given intravenously in a large initial dose of 5 gm. and daily intravenous doses of 2 gm. should be continued with accurate blood and cerebrospinal fluid sulfadiazine level determinations. It is essential that the fluid intake of the patient be kept at a high level, not below 3000 cc. in twenty-four hours, and of this total 1000 cc. should consist of one-sixth molar sodium lactate solution because it has been shown that sulfadiazine is more effective when alkalinity as measured by the reaction of the urine is maintained. Therapy should be continued for some time after the temperature has reached normal and symptoms have subsided.

Experience in the treatment of meningitis and ventriculitis with penicillin by Cairns, Duthie, Lewin and Smith[1] has shown that when a dose of 4000 to 10,000 units is injected into the lumbar subarachnoid space or the lateral ventricle it spreads, in the absence of block, to all parts of the cerebrospinal fluid pathways. In most patients it persists in the cerebrospinal fluid at a level above that required for complete bacteriostasis for about twenty-four hours Although a primary pyogenic meningitis can be treated satisfactorily by daily subarachnoid injections of penicillin, this method alone is useless when the meningitis is secondary to a brain abscess or any other focus of suppuration. The latter must also be treated surgically and effectively

Cairns has emphasized that an adequate concentration of penicillin must be maintained in the cerebrospinal fluid until the clinical signs of meningitis have disappeared and the cell content of the fluid has

[1] Cairns, H Duthie, E S, Lewin W S, and Smith, H V "Pneumococcal Meningitis Treated With Penicillin," Lancet, May 20, 655, 1944

fallen. Otherwise the meningitis may suddenly recur, a block in the subarachnoid space may develop and further injections are ineffective. It should also be emphasized that it is necessary to have an opening in the dura mater over the cerebral hemispheres before any substance injected into the subarachnoid space will diffuse freely throughout the cerebral subarachnoid spaces.

It is not difficult to establish and maintain an adequate level of sulfadiazine in the cerebrospinal fluid by oral or intravenous administration. To obtain the same result with penicillin it is necessary to inject it into the cerebrospinal fluid pathways under strict aseptic precautions at not less than twenty-four-hour intervals and with regularity. Though the sulfonamides may be less effective than penicillin in some types of meningitis, notably pneumococcic, the ease of administration and passage through the blood-cerebrospinal fluid barrier makes them invaluable in the treatment of this once dreaded complication of a craniocerebral injury.

Brain Abscess.—Abscesses may develop in the tract of the brain injury and are under those circumstances commonly associated with a meningitis. On the other hand, an abscess may occur following an apparently trivial scalp and skull injury, but one which has produced a small tear in the dura mater.

In one patient, struck upon the head during a hold-up, a brain abscess was found during the course of an operation for what had been diagnosed as a chronic subdural hematoma. Hemiparetic symptoms had been slow in their onset with inconstant variations which seemed pathognomonic of a hematoma. Greatly to our surprise we found that the original wound had been produced by a knife blade and the skull had been penetrated. The dura had been torn but slightly, yet the abscess was localized immediately beneath this stab wound.

The importance of removing indriven fragments of bone in the prevention of a brain abscess following a craniocerebral injury has already been emphasized. If an abscess develops in the first few weeks following a brain wound, it should be treated by removal of the foreign fragments, and local drainage of the entire abscess cavity. Sterilization of the cavity can be accomplished by the intensive use of systemic antibiotic and chemotherapy, depending directly upon the isolation and identification of the organisms. It must be remembered that the thorough, meticulous surgical treatment of the original wound is the best way to prevent a cerebral abscess.

Pneumocephalus or Aërocele.—A fracture of the skull which passes through the paranasal sinuses or mastoid air cells may be accompanied by air within the cranial cavity. Sneezing, coughing, straining, or swallowing is necessary to force air through the bony

and dural defect into the cranial chamber. The air may be located within the subarachnoid spaces, the subdural space, the brain, or the ventricles. Air may often fill the frontal lobe and extend backward into the parietal lobe. The shadow is usually round or oval and projects backward from the frontal lobe. The cavity always communicates with the frontal or ethmoid sinus by an opening which may be so minute as to be overlooked easily. The majority of fractures which involve the paranasal sinuses are asymptomatic and should be left alone. Intranasal douches are strictly contraindicated,



Fig. 44 —Aërocele following fracture of the skull.

and the patient should not be permitted to blow his nose violently Compound fractures of the frontal sinus should be operated upon and the posterior wall of the sinus inspected carefully. If the dura is torn, it should be repaired immediately. The symptoms of aerocele are in the main those of increased intracranial pressure and often the patient complains of a moving object inside his head. A cerebrospinal fluid discharge may be present and rhinorrhea after sneezing or a change in posture is almost pathognomonic. Roentgen-ray

films will furnish an absolute diagnosis. Dandy[1] reported 28 cases of aërocele and has suggested an operative method by which a transplant of fascia lata is used to repair the dural tear. (Fig. 44.)

Post-traumatic Symptoms.—The most common complaint of patients who have apparently recovered from a skull injury is headache. The majority of patients complain of generalized pains in the head which are not well localized and which are increased by changes in posture. A small number complain of well-localized headaches which are persistent and unchanging in their location. Penfield[2] has shown that many in the second group of patients have a collection of fluid in a localized portion of the cerebral subarachnoid spaces. In his opinion this accounts for their severe headaches. He has provided relief for these patients by substituting air, introduced by lumbar puncture, for the fluid. The air is later absorbed and supposedly the normal intracranial mechanics are re-established. We have not observed the amount of benefit from this procedure which Penfield describes though definite encephalographic changes were demonstrable.

Dizziness, which must be differentiated from a true vertigo, is also a common complaint. Upon close questioning of the patient, it may be developed that the complaint is really of unsteadiness upon stooping or looking upward. True vertigo, in which objects rotate about the patient or *vice versa*, may follow a basilar skull fracture in which the eighth cranial nerve is injured.

Opinions have varied for many years concerning the late symptoms of craniocerebral injuries between the organic and functional points of view. Strauss and Savitsky[3] have insisted that the subjective symptoms of the post-traumatic syndrome, consisting of headaches, dizziness, unusual fatigue upon effort and vasomotor instability, are dependent upon a disturbance in intracranial equilibrium due directly to the blow on the head. Others[4] have called attention to the fact that the neuroses which follow injury to the head do not differ greatly from those which occur after injury to other parts of the body in which injury to the central nervous system is not in question.

The differences between the two opinions is perhaps one only of emphasis, because common factors are recognized by both. The

[1] Dandy, W. E.: Pneumocephalus (Intracranial Pneumatocele or Aërocele), Arch. Surg., 12, 949, 1926.

[2] Penfield, W. Meningocerebral Adhesions; a Histological Study of the Results of Cerebral Incision and Cranioplasty, Surg., Gynec. and Obst., 39, 803, 1924.

[3] Strauss, S., and Savitsky, N.: Head Injury, Arch. Neurol. and Psychiat., 31, 893, 1934

[4] Hall, G. W., and Mackay, R. P.: The Posttraumatic Neuroses, Jour. Am. Med Assn., 102, 510, 1934.

question seems to be whether injury to the head produces organic disturbances of the central nervous system with resulting so-called functional symptoms or whether it acts as the exciting factor in a person already prone to a neurosis.

Guttmann and Horder[1] have reported upon sixty cases of craniocerebral injury in children. In the acute stage emotional symptoms were more impressive than clouded consciousness and intellectual loss. Post-traumatic symptoms were observed in 10 percent of the patients and their incidence depended to a large extent upon environmental factors. Irritability was the most common symptom. Persistent behavior disorders were rare as also was intellectual impairment.

The diagnosis from organic diseases is usually not difficult. The main difficulty is met with in differentiating the neuroses from *malingering. In the neurosis there is a period of meditation between* the injury and the appearance of the neurosis. This may be days, months, or years. The symptoms and signs met with are insomnia, fatigue, exaggerated tendon reflexes, increased mechanical excitability of muscles and nerves, abnormal irritability of the cardiovascular system, and tremors. The ease with which fatigue is induced in both the mental and physical spheres is one of the most notable features. On the psychic side the patient may present phobias, hypochondriasis, introspection, preoccupation, headaches, giddiness, cerebral congestion, and intolerance of alcohol.

The neurotic deceives himself, often others also, but usually unconsciously and without design. The malingerer deceives others, *not himself, consciously and with intent, and always with definite* motives. The neurotic is given to extravagant and bizarre comparisons and unconsciously tends to exaggerate the symptoms. The malingerer is often sullen, suspicious, and ill at ease. He is conscious of the unreality of his symptoms and is more careful than the neurotic in choosing his words to avoid being enmeshed.

Residual symptoms due to organic lesions of the central nervous system depend entirely upon the extent and location of the injury Hemiplegia, or a monoplegia, may occur, but if there has not been an actual destruction of brain tissue, the degree of recovery may be most striking Daily physical therapy treatments consisting of massage, passive movements, electricity, and active exercises should be employed to advantage in these patients *Facial paralysis,* peripheral in type, may be the result of an injury to that nerve in its course through the facial canal. The physiological function of this nerve may be interrupted by hemorrhage within the facial canal,

[1] Guttmann, E and Horder, H Head Injuries in Children and their After-Effects; Arch. Diseases in Childhood, 18, 139, 1943.

and under such circumstances, aided by a supporting splint for the facial muscles and physical therapy, the degree of recovery may be striking. The optic, oculomotor, trochlear, and abducens nerves may be permanently injured if the fracture line extends across the base of the skull and injures these cranial nerves in their course. Residual sensory symptoms from cortical damage are rarely encountered. Aphasia may occur as a part of a hemiplegia, but unless the cortical damage is enormous, recovery may be expected. Mental symptoms may persist for a long period of time following a skull injury. They may consist of disorientation, loss of memory, emotional and habit changes. One such patient was disoriented, and uncoöperative, for eight weeks following an accident in which the most severe external injury was a laceration of the scalp. There were no symptoms of a localized lesion of the nervous system and the patient had regained consciousness within four hours after the injury. It was necessary to resort to intranasal feedings during the time that every possible complicating factor was investigated. Rather suddenly, the patient became rational, bright, and within a week was apparently quite normal, presenting an entirely different personality from the patient we had agonized over for weeks.

Physical and mental rehabilitation of patients who have received a craniocerebral injury requires good judgment and skill. Psychotherapy is as important as physical therapy. The patient should be encouraged in every way to think that the injury has been only a slight one. Terms like "concussion of the brain" and "compound fracture of the skull" must be absolutely forbidden and in the presence of the patient such simple terms as "bump on the head" and "cut on the head" substituted. Lumbar punctures upon conscious patients are usually unnecessary and should be avoided because they add little useful knowledge, have little therapeutic effect and often leave a deep and bad psychological scar. An arbitrary period of bed rest for two to three weeks, as was formerly practised, has been proven from experiences with wounded soldiers to be poor therapy. Instead, as soon as the patient feels able to sit up or go to the lavatory, or to feed himself, he should be allowed to do so. He should be encouraged to take part in the care of his own person and surroundings.

Epileptiform seizures, which usually begin as focal attacks, may follow immediately upon the receipt of a cerebral injury or their onset may be long delayed. In the majority of cases, convulsions *do not occur*. It is impossible to prognosticate the occurrence of convulsions upon the location or extent of the brain injury. The basic status of the patient's nervous system probably plays a large factor in their development. In those cases of definite focal attacks

which are followed by motor impairment, plastic cranial operations may be of service. We have had occasion to operate upon a patient who had suffered a cranial injury twenty years previously. She was seriously ill at the time of her accident but eventually recovered. During the intervening years she had been a social service worker with many executive duties. In the last four years she had developed increasingly frequent Jacksonian attacks each followed by definite motor weakness in the right side. Her roentgen-ray films showed an old fracture of the skull in the left temporal bone.

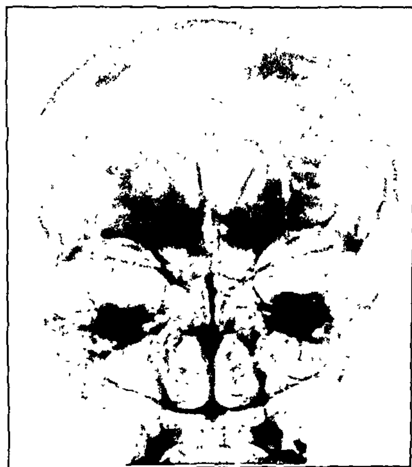


FIG 45.—Encephalogram showing dilatation of ventricle on side of the old depressed fracture of the skull Both ventricles are pulled toward the left side.

Encephalograms demonstrated a mechanical deformity in the ventricular system, in which the ventricles were pulled to the side of the original injury. At operation the dura mater and cortex were adherent to the lines of fracture in the temporal bone. A small cyst of the cortex had formed at the site of the injury. This was removed, and the adherent bands divided, allowing the brain to resume its

normal position. During the nine years following operation the patient has had no recurrence of her convulsive attacks. (Fig. 45.) This result was perhaps more favorable than in the majority of patients who may require carefully controlled anti-convulsant therapy with bromides, phenobarbital, or dilantin sodium.

Tantalum and acrylic resin plates were used extensively in the repair of cranial defects resulting from severe injuries received during the World War II. There is no doubt about the effectiveness with which they restore the symmetry of the skull and the patient's self-confidence. It is to be hoped that the prevention of adhesions between the brain, its dura mater and the scalp will effectively deter the onset of epileptiform seizures in the patient. A careful, analytical study of these patients after many years following their injury will be necessary to settle this point satisfactorily.

Tantalum is by far the easiest material to process and fit at the time of operation to repair a skull defect. In patients who develop epilepsy, however, the translucent acrylic resin plates permit subsequent studies of the ventricular cavities and subarachnoid spaces. It is wiser not to attempt a primary repair after craniocerebral injury. Two months should elapse if the wound heals by first intention. If there has been an initial infection, repair should be postponed for six months or longer.

In spite of an attempt to avoid speaking dogmatically of the treatment of craniocerebral injuries, I find that a description has been given of our own procedure in many instances where the custom of others may be recognized immediately as another method of accomplishing the same result. The important thing is to avoid complexity in therapy. The greater number will recover if they are regarded and treated as an individual problem. The known physiological, pathological, and therapeutic facts must be fitted to the patient. It should not be a question, for example, of always using lumbar puncture drainage to the exclusion of hypertonic solutions, or *vice versa*. There are definite indications for each therapeutic step, and when the indication does not exist, the therapy usually fails to accomplish its purpose.

CHAPTER III

INTRACRANIAL TUMORS

"Neurological surgery is a large subject and the surgery of brain tumours is a special field within it Indeed, the day may well enough come when certain surgeons will find enough to keep themselves fully occupied by attending exclusively to tumours of a single type."

HARVEY CUSHING.

THE progress of the surgical treatment of intracranial tumors has been associated intimately with the advancement of asepsis and surgical technique in general, methods of more accurate diagnosis, and a correlation of the pathology of tumors encountered with the clinical course of the patient. It is a long way from the early trepanations of Jaboulay to the present surgical technique of an osteoplastic craniotomy. It was Cushing who first insisted upon the necessity of observing the meticulous details of strict hemostasis and asepsis for success in intracranial operations. The introduction of silver clips to ligate bleeding cortical vessels; the use of cotton pledgets moistened in saline solution for sponges; the suction apparatus, so useful in keeping the field of operation well defined; the employment of an electrosurgical unit for transection of the cerebral cortex and the removal of large vascular growths; the accurate closure of wounds layer by layer with the finest suture material; the recent development of fibrin foam and gelatin sponge for complete hemostasis, all of these contributions of neurological surgeons have had their cumulative influence upon all fields of surgery.

Closely associated with these more or less technical matters, and in fact by their nature a challenge to the mechanical side of neurological surgery, have been the contributions to the art of the diagnosis, localization and histopathology of intracranial growths. Physiological experiments have added to our knowledge of the function of the various parts of the brain and to the mechanics of the cerebrospinal fluid pathway The ophthalmoscope and perimeter, the *diagnostic instruments of a branch of medicine distinct from neurology* and surgery, also have made it possible to localize intracranial tumors more accurately and earlier in their course More recently, experimental and clinical investigations of alterations in the normal waves of cerebral activity by means of the electro-encephalograph *have yielded results which will prove to be of real diagnostic aid.*

The contributions of the science of roentgenology to neurological surgery have been innumerable. The correct interpretation of the

direct evidences in the skull of various intracranial neoplasms is not only possible, but the corroboration of the probable pathology to be encountered at operation is now a common occurrence. When one adds to this the visualization by ventriculography and encephalography of the deformities produced by tumors, the dependence which neurological surgery places upon roentgenology may be realized.

Most recently, the results of atomic research have added another diagnostic method to aid in the solution of intracranial neurological problems. It has been shown that the concentration of a radioactive isotope dye in intracranial tumor tissue can be detected and outlined by external examination of the skull with a Geiger-Mueller counter. What appears to be a matter of even more importance is the ability to identify infiltrative tumor tissue at the operating table by a surgical probe counter.

Finally, all of these forward steps in matters surgical and diagnostic would be of little avail were it not for the progress which is being made in the correlation of the pathology of the lesion and the clinical history and course of the patient. Today a diagnosis of an intracranial tumor in a given location is not enough. More often than not, the surgeon is cognizant of the probable pathological nature of the lesion which he is to attack.

However, the average practitioner of medicine has a pessimistic attitude toward the recovery of a patient with an intracranial tumor. In practically every instance this is because he has not been acquainted with a few fundamental facts concerning the diagnosis and treatment of intracranial tumors. He is likely to group them all as perfectly hopeless and this is far from the true facts of the matter.

It is difficult to reduce a subject of such an enormous scope to fundamentals which will afford help to the physician who encounters an intracranial tumor only occasionally. This is more forcibly realized when one sees the monographs which have been written upon each single group of brain tumors, but these monographs primarily concern those devoted to the practice of neurology and neurological surgery.

The time has passed when intracranial tumors may be discussed as a whole. They are composed of a great diversity of neoplastic types. In addition, tumors in certain areas of the brain produce different localizing symptoms, to say nothing of the fact that the various types of tumors produce different symptom complexes in the same location. The neurological surgeon should be able to diagnose the presence and location of a tumor and, in addition, its pathological type. Verification of such a diagnosis at operation offers valuable aid in formulating a prognosis and intelligently directing the postoperative treatment. It becomes necessary, there-

fore, to answer three questions concerning the patient's symptoms. First, are they the symptoms of an upper motor neuron lesion and, if so, are they due to an intracranial lesion? Second, are the symptoms due to an intracranial tumor and third, what kind of a tumor is it likely to be? To answer these questions, a knowledge of the anatomy and physiology of the nervous system and familiarity with the histological characteristics and nature of growth of the various tumor types are necessary. To be able to answer the first two questions will mean that the patient will receive sound advice and the answer to the third question will be an amplification of that advice which can well be supplied by the neurologist or neurological surgeon.

CLASSIFICATION OF TUMORS

A great deal has been added to our knowledge of intracranial tumors because neurological surgeons have religiously attempted to verify the tumor microscopically, rather than being satisfied to base their opinion of its nature solely upon a gross examination at operation. As a result, accurate information is available concerning the clinical behavior of the various types of tumors before and after operation, or other types of therapy. It is at once obvious that the varying types of tumors have totally different clinical characteristics dependent upon their histological character. More recently, it has become more and more clear that among the large group of tumors, classed as gliomas, there is a great disparity in behavior. As a result, efforts have been made to classify intracranial tumors histologically and in particular, the gliomas.

THE GLIOMAS

Approximately 50 per cent of all intracranial neoplasms are in the *glioma* group. These tumors, which are primary tumors of the brain substance, have been subdivided by various methods by many individuals, and as a result there is still no completely uniform nomenclature by which the gliomas may be classified.

These growths arise in various regions of the brain and they may be composed of adult cells, embryonic cells, or a mixture of the two. Since there is such a structural variability in the gliomas it is perhaps most logical and direct to classify them according to their histogenesis This may give a clue as to their growth history and indicate their degree of malignancy. Correlation of the clinical manifestations and histopathology of each group of tumors has made it possible to learn more about their course, if untreated; the results of surgical treatment, and their reaction to roentgen-ray and radium therapy

VERIFIED INTRACRANIAL TUMORS
January 1, 1952

	Total	Percent
Gliomas	644	53 0
Glioblastoma Multiforme	331	
Astrocytomas	191	
Astroblastomas	6	
Oligodendrogliomas	30	
Spongioblastomas Unipolari	10	
Medulloblastomas	37	
Ependymomas	24	
Papilloma Choroid Plexus	7	
Pinealoma	5	
Ganglioneuroma	3	
Neuroblastoma	0	
Medullo-epithelioma	0	
Neuro-epithelioma	0	
Meningiomas	177	14 5
Tumors Hypophysis	89	7 3
Chromophobe Adenoma	68	
Chromophile Adenoma	17	
Adenocarcinoma	4	
Tumors of Blood Vessels	63	5 1
Hemangioblastoma	38	
Angioma	25	
Acoustic Neurinoma	74	6 0
Congenital Tumors	34	2 7
Craniopharyngioma	22	
Chordoma	2	
Dermoid	6	
Teratoma	1	
Cholesteatoma	3	
Metastatic Tumors	111	9 1
Carcinoma	93	
Sarcoma	11	
Hypernephroma	7	
Granuloma	5	0 41
Tuberculoma	4	
Gumma	1	
Miscellaneous Tumors	10	0 82
Cysts (various)	4	
Osteoma	4	
Osteochondroma	1	
Meningeal Sarcomatous	1	
Unclassified Tumors	8	0 65
Total	1,215	

A simple method of recalling the development of the gliomas according to their cell types is to refer to the schema by Bailey. (Fig. 46.) This shows the growth history of the various descendants of the cells of the primitive medullary epithelium, and the probable order in which the gliomas arise from embryonic to adult cells, with corresponding degrees of malignancy. Every step in the various paths of growth cannot actually be demonstrated, but from much accumulated evidence it is apparent that this schema of histogenesis

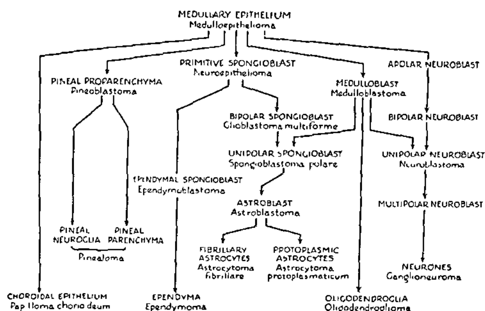


FIG. 46.—Diagram of the embryological development of cell types of the central nervous system and the tumors related to them. (According to the schema of Bailey.)

offers a reasonable working hypothesis which is supported by many investigators by detailed studies of the staining properties, the appearance of fresh smears of tissue, the cytological characteristics, the gross appearance of the tumors, and the clinical histories of the patients. Reference to this chart later in the chapter will substantiate the statement that in ascending from the primitive to the adult cell type there is a corresponding change from the malignant to the relatively benign gliomas.

Glioblastoma Multiforme.—The tumors known as *glioblastoma multiforme* (sometimes referred to as "spongioblastoma multiforme") belong to the group of the more malignant gliomas of the brain. There have been 331 of these highly malignant and rapidly growing tumors in our series and they have occurred without exception in the cerebral hemispheres of adult patients. They constitute about 50 per cent of all the gliomas, and because of their malignant nature they may erroneously represent to the average clinician the

typical story of all intracranial tumors. It is this inaccurate belief which frequently influences the physician to assume a pessimistic attitude, and his discouraging advice to his patients or their relatives may be completely groundless. The clinical story of these tumors must be contrasted with the more favorable, and many times brilliant, results obtained in the surgical treatment of other intracranial tumors.

The glioblastomas very commonly have a dramatic and sudden onset which may be misleading to one with the conception that all intracranial tumors have a slowly progressive course. They tend to originate in the central white matter of the cerebral hemispheres, but do not always originate so deeply, and they frequently extend into the cortex, greatly widening and distorting the convolutions of the hemisphere, and present themselves boldly on the surface of the brain. At times the tumor may appear to be so superficially located and so well circumscribed that it would seem possible to enucleate it wholly. But more often these tumors do not present themselves on the surface of the cortex, but only give a surface manifestation of their underlying location, as seen in the wide, flat gyri, the soft, yellow-gray cortex, the shift in the normal position of the surface veins and arteries, and a brain under such tension that it tends to herniate out through the operative craniotomy. Beneath such an area a large glioblastoma may lie, and as it grows in all directions from its original locus it destroys and infiltrates the surrounding brain in much the same manner as a bit of rot enlarges and eventually destroys an apple. Grossly, there may not be the slightest visible line of demarcation. Such tumors may show a predilection for the large fiber tracts which pass from hemisphere to hemisphere and, spreading across the corpus callosum to the opposite side, they may involve the opposite side with the production of certain symptoms of mental change which facilitate the clinical diagnosis of such an extension. The glioblastomas tend to undergo degenerative changes, and grossly they may present many hemorrhagic foci, yellow areas of degeneration, numerous large and small cysts, and more solid reddish-brown masses of undegenerated tumor tissue.

The frequent rapid onset of symptoms in these tumors may be so pronounced as to cause a sudden coma and paralysis. It is this group of neoplasms which is often mistaken for a vascular disease. The patient usually survives this primary serious insult and recovers from his coma, only to show a mental dullness and stupor which lightens and deepens intermittently. A high degree of brain edema and increased intracranial pressure occurs very quickly and a high-grade papilledema with hemorrhages in the retina is quite typical. Often a glioblastoma in the frontal lobe may show a degree of mental hebetude out of all proportion to other physical symptoms.

A professor of Greek left the city with his family for a vacation and, after driving his automobile for four days, he set about putting the camp in order. The next evening he complained of a severe headache, and within a few hours became comatose. He remained so for three days and just as suddenly became alert and oriented. He was then brought back to his home and was taken to a hospital where he gradually became stuporous, though he could be aroused for short periods to take food. When external stimuli ceased, he would immediately fall into a deep sleep.

On examination, this patient showed a slight, but definite, weakness of the left side of his face and the left upper extremity would waver and fall away during tests for motor function. Intravenous administration of hypertonic solutions would be followed by brightness and mental alertness of an almost unbelievable degree when compared with his previous states.

At operation a large glioblastoma was found in the temporal and frontal lobes. It was so soft that the tumor was removed by suction.

Although the average survival period from the onset of symptoms for these tumors is about twelve months, and there may be only six or nine months before there are signs of recurrence after operation, this respite is appreciated by the patient and his relatives. In several patients two and even three operations have extended life to a period of over two years, during which time the patients were not only more comfortable than they had been, but were able to fulfill both business and social obligations, a fact which was a great comfort to them.

Although the glioblastomas are infiltrating, it is the custom of the majority of neurological surgeons to attack these tumors with every possible surgical means at their disposal. The electrosurgical unit has made it easier to block out a mass of tissue and then to remove the remaining tumor by scalloping with a loop electrode. Often the center of the tumor mass is soft and almost gelatinous and liquid so that it may be removed with a suction apparatus. Recently, we have been fortunate to encounter glioblastomas in the frontal lobe of the less dominant hemisphere in several patients and have been able to remove large portions of the lobe. In our group of patients of this type we have removed portions of the lobe and tumor weighing from 60 to 150 gm. with immediate clinical results which promise much. (Fig. 47.)

It is not for us as surgeons to give up all hope for the alleviation of these tumors until the end is at hand. It has been this attitude of utter hopelessness which has cheated many patients with other types of intracranial tumors out of their chances to be well. It has also tended to discourage attempts to deal in other ways with these admittedly difficult tumors, and certainly the attitude of the defeatist will not lead to a solution of the problem which they present.

The use of a Geiger-Mueller surgical probe counter during an operation upon a glioma aids materially in obtaining as complete removal

of the tumor as is possible. The difference in counts per minute in tumor tissue, where the radioactive isotope is highly concentrated, and normal brain tissue is striking.

It is necessary, in addition to removing the tumor as completely as possible, to provide a large decompression opening in the skull by removing the lower two-thirds of the bone flap or by taking it out entirely. This affords relief from pressure in the event of recurrence. To decompress these tumors and not effect a removal, which may be incomplete it is true, is to look forward to an early fatality. This is well illustrated in the following case in which the growth was not disclosed at an operation, performed elsewhere, and the diagnosis upon admission to our clinic was an "unverified tumor."



FIG. 47.—Illustrating the use of the electrosurgical unit in the removal of gliomas (*a*) coagulation of surface vessels with the ball electrode; (*b*) incision of the cortex with the flat blade electrode; (*c*) removal of the tumor with the loop electrode.

A hard-working farmer woman, aged thirty years, suddenly developed severe headaches and vomiting only five weeks before admission to a hospital. She continued to do her work until she was found unconscious on the ground near her kitchen door. She was taken to a hospital where a high-grade papilledema was recognized and a right subtemporal decompression was performed. Her headaches promptly disappeared and she was able to return to her home. Within a month the patient was admitted to our clinic with a left hemiplegia, in coma, with a papilledema of 5 diopters, and a bulging decompression area.

An osteoplastic craniotomy was performed and a large glioblastoma in the right temporal lobe was removed with the elctrosurgical unit and by suction. The bone flap was removed and a large decompression afforded. The patient made a good recovery, her papilledema subsided completely, and she was given physical therapy treatments to aid in recovery of the function of her arm and leg. In spite of deep roentgen-ray therapy which was administered intensively, she succumbed within eight months.

Glioblastomas are characterized by their highly polymorphous microscopic appearance. The predominating cell is the altered, neoplastic neuroglial cell, but occasionally in the newly-invaded, less degenerated areas, remnants of the true neural elements and ganglion

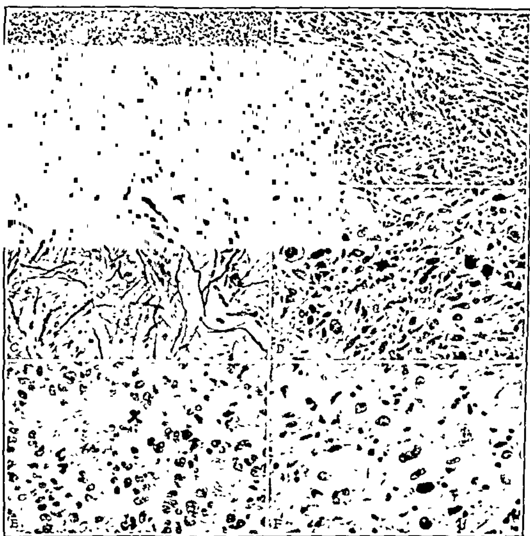


FIG. 48 — Photomicrographs of six different types of glioblastoma multiforme (A) low-power magnification to show general architecture of a glioblastoma, × 110. (B) glioblastoma showing predominance of spongioblasts. × 200, (C) glioblastoma stained by Cajal's method showing unipolar spongioblasts × 470, (D) glioblastoma showing mitotic figures, × 375, (E) glioblastoma showing a large predominance of protoplasmic astrocytes, × 465, (F) glioblastoma showing giant cells with hyalinization of cytoplasm, × 465.

cells may persist The nuclei vary greatly in distribution, size, shape, and chromatin material and the amount of cytoplasm surrounding the nuclei may be extremely scant or it may be fairly abundant. Hyalinized cells, or entire areas of hyalinization, are common Mitotic figures are to be seen everywhere, and many of

them are abnormally shaped. There is no characteristic architectural arrangement in the glioblastomas. One may find areas of densely packed cells with no particular arrangement, while neighboring areas may show cells closely packed into pseudo-palisade arrangement. Multinucleated giant cells may stand out conspicuously in a background of fairly uniformly shaped neoplastic glial cells. Areas of frank cystic necrosis may be contrasted to other relatively acellular areas where a fibroblastic repair has been accomplished. Blood-vessels frequently are very numerous, and their walls may be thickened, hyalinized, or even ruptured. Foci of interstitial hemorrhage due to such blood-vessel breakdown are common. A study of blocks of tissue from various parts of the tumor often shows a great variation in the microscopic appearance in the same tumor. At the periphery, where the vascular supply is still intact and the tissue is relatively solid, one may see the proliferation and invasion of the tumor cells into the frontiers of normal brain tissue. (Fig. 48.)

We have compared a series of these patients who have not received deep roentgen-ray therapy after operation with those who have. Many times the immediate benefits following operation and roentgen-ray therapy have been so striking that we have felt greatly encouraged, only to realize that actually the clinical course ran on unchanged But it is to be remembered that even the glioblastomas vary in their degrees of malignancy, and even though the tumor may not have been removed widely it is always possible that such follow-up roentgen-ray therapy will have a beneficial effect.[1] Especially in those patients where all visible evidence of tumor has been removed at operation one should give such therapy and feel justified in expecting a longer postoperative survival period. We have subjected a group to treatment with a radium bomb and in a few patients, we have implanted radium needles into the tumor mass, leaving them for as long as two weeks. The results of irradiation by these methods have not been encouraging. Irradiation must be delivered directly to the bed of a tumor surgically removed as completely as possible, or to a surgically inoperable tumor. Experimental and clinical studies which show that radioactive gold, or phosphorus, can be injected into tumor tissue for their irradiation effect without damaging normal cerebral tissue are hopeful steps toward obtaining more satisfactory results in the treatment of these tumors.

[1] In 4 cases of glioblastoma multiforme which have been treated with roentgen-rays following operation, the central area of necrosis was more extensive and the overgrowth of collagenous connective tissue was more abundant than in 2 untreated control cases. However, the growth of the tumor at the periphery had not been arrested A study of a larger series of such tumors should decide the question whether the difference in the age of the tumors of the treated and untreated cases determined the difference in the histological picture or whether it was produced by the roentgen-ray therapy.

This group of gliomas provides the greatest challenge to neurological surgery. Progress cannot be made by adopting the surgical philosophy of identification of the tumor by a biopsy section obtained through a burr hole opening and refusing further surgical steps. The same philosophy obtains in those clinics where from the history and examination, it is concluded that the tumor is a glioblastoma multiforme and the patient is refused entrance to the hospital so that another may be admitted whom it is thought has an intracranial tumor more favorable for surgical removal.

The surgical mortality is high;[1] the lesion may require more than one operation; the tumor may be so situated that a surgical attack would result in a vegetating, helpless individual; the results judged upon restoring the patient to his previous social and economic position are discouraging, but our present methods of surgical attack are comparatively crude and must be improved. It may be possible to develop the use of radioactive isotopes, with the proper half-life values, which may successfully cause these tumor cells to degenerate and be followed by a minimal neurological defect. Early recognition of the signs of impaired cerebral function, an imaginative and hopeful surgical approach, and an active follow-up record system will eventually make their mark upon a difficult problem.

Astrocytoma.—The *astrocytoma,* though not a benign tumor, is among the less malignant and slower growing of the glioma group. In Cushing's large series of verified tumor cases, it was found that the average survival period from the time of onset of symptoms in this group was well over six years. They are found predominantly in the cerebral hemispheres of adults and the cerebellar hemispheres of children. In either location, but especially the latter, they may be cystic. They are, like other gliomas to be described, made up of adult glial cells but their clinical manifestations are not so much dependent on the type of astrocyte as upon the location of the tumor in the brain.

Cerebellar Astrocytoma.—Astrocytomas of the cerebellum often are found within a lateral lobe rather than in the midline and, as stated, are frequently cystic. These cysts may be of two types In one, the walls of the cavity are composed entirely of a thick layer of tumor tissue and it is probable that the cyst occurs as the result of necrosis and liquefaction of tumor cells, this in turn being followed by a transudation of serum into the area of degeneration. The second type, and that which is more commonly found, consists of a cystic cavity lined with a smooth, thin, velvety tissue which is

[1] Every death in the hospital following an operation, regardless of the cause and the length of the interval, is recorded as a postoperative fatality Many Veterans Administration Hospital patients remain in hospital for years as convalescents.

cerebellar and not tumorous. In one portion of the wall there will always be found a small, dark, meaty nodule of tumor which projects into the cavity. It is probable that in this type there is a transudation of serum from the blood-vessels on the surface of the nodule of tumor tissue. The fluid of these cysts varies from a light, clear, canary-yellow to a dark, dirty, brown color and because of its high protein content it clots quickly upon standing.

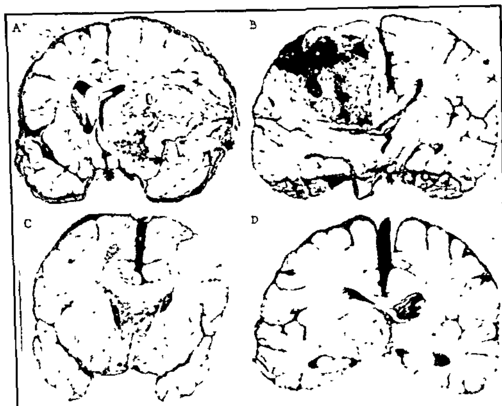


FIG. 49.—Gross appearance of [illegible]

Clinically, it should not be assumed that these are tumors which occur only in children, because in our own series they have been found more frequently in young adults. The important fact is that an astrocytoma which occurs in childhood is predominantly a cystic one and the progression of symptoms may be very slow and insidious. Not infrequently, particularly in children, the first symptoms consist of early morning headaches and vomiting which may lead the unwary into persistent and erroneous investigations of the gastro-intestinal tract. Difficulty in walking and awkwardness in the use of the upper extremities may be the initial symptoms in older indi-

viduals. As the tumor grows and the cyst increases in size, intracranial pressure rises with an accompanying failure of vision, diplopia, and perhaps other signs of pressure upon the fifth, seventh, ninth and tenth cranial nerves on the same side. Later, pressure upon the pons and medulla give rise to motor and sensory symptoms and the coarse, dysmetric nystagmus of cerebellar dysfunction develops.

The insidious onset of the symptoms of a cerebellar astrocytoma is well illustrated in three of our patients.

A young, married woman's only complaint was persistent vomiting which occurred in attacks during which she was unable to turn her head from the right side. There was no complaint of headache and when the vomiting attack subsided, usually within forty-eight hours, she was able to go on with her housework without the slightest difficulty. These attacks of vomiting increased in frequency and lasted longer so that it was assumed that she must have a gastro-intestinal dysfunction.

However, it was thought strange that she could not turn her head upon the pillow without vomiting and she was accused of being an hysterical young woman who had a chronic appendicitis. Finally, it was found that quite often she had a very pronounced nystagmus, and after one bout of vomiting she had an internal squint in the right eye.

After twelve months of doubt and indecision, she was referred for observation and there was a weakness of the right external rectus muscle; a nystagmus with coarse movements as she fixed her eyes toward the right; an awkwardness in the use of her right hand; and a tendency to deviate toward the right as she walked. Upon questioning her carefully, it was found that when she had her headaches, they were suboccipital in location, and her neck muscles became very tender. As a matter of fact, she carried her occiput tilted toward the right.

There was no papilledema, but upon these signs and symptoms alone, a suboccipital craniotomy was performed and a cyst was found in the dorsolateral portion of the right cerebellar hemisphere The mural tumor nodule was about the size of an olive and projected into a cavity which held 45 cc. of yellow, viscous fluid The patient remains perfectly well sixteen years after operation.

A girl of ten years had been entirely well until three months prior to admission to the hospital, at which time she began to complain of suboccipital headaches of increasing severity. For two months there had been daily vomiting, and for six weeks she had complained of a staggering gait and of falling with no tendency to fall to one particular side. There was no loss of visual acuity, no diplopia and no papilledema. Three weeks before operation she was given a short course of deep roentgen-ray therapy, following which there was a temporary relief of her symptoms

At operation, a cyst was found within the left cerebellar hemisphere It was lined by a velvet-like membrane and attached to one wall was an almond-sized nodule of dark red tumor tissue. The cyst was evacuated of its yellow, rapidly-clotting fluid and the child returned to school Now, fourteen years later, she is entirely free of all symptoms and there is no evidence of any cerebellar dysfunction

A young accountant noted that he could not write well and he became more and more disturbed at the appearance of his figures. He did have an occasional headache which was severe, but it was attributed to some indiscretion in diet or to his confining hard work. The deciding factor which sent him for observation was that his employers thought he had come to work intoxicated when they saw his staggering, ataxic gait. A large astrocytomatous cyst with a mural nodule was removed in this case, although at no time was a measurable papilledema present. The patient remains perfectly well and at work seventeen years following operation.

It is among patients with cerebellar astrocytomas that one will find the occasional history of an operation many years before when the practice was to empty the cystic cavity by simple needle puncture and do nothing else. It was not then known that there was a tumor nodule in the wall or that the entire wall consisted of tumor tissue which would continue to refill the cavity. Yet, in spite of this defect in knowledge and treatment, the patients did well because the tumors were slow in growth. We had occasion to operate upon one such patient whose cyst had been emptied fifteen years previously and nothing more had been done. At the second operation the tumor nodule, a large, firm, purple mass, was completely removed. Now, twenty-four years later, the patient is at work, an economic and a socially independent individual, thirty-nine years after his tumor was first diagnosed. In such tumors, then, the entire wall of tumor tissue or the nodule must be completely removed to effect a permanent cure.

As Cushing has pointed out, a surgeon's case mortality statistics will improve with experience and particularly with the increase in knowledge which has followed a careful and detailed study of the histological and clinical characteristics of the tumors removed. Cushing's own large series of 91 astrocytomatous cysts of the cerebellum was a demonstration of this fact. Up until 1930, he had performed 113 operations upon 76 patients, due chiefly to recurrences and owing to a lack of a complete understanding of the life history of these tumors. There were 15 fatalities which produced an operative mortality of 13.3 per cent and a case-mortality of 19.7 per cent. However, in the three-year period from 1928 to 1931, 34 operations were performed upon 29 cases in which the tumor had been wholly removed from the cyst wall, and there was only one death. In the last five-year period, therefore, his case-mortality for this group of tumors was 3.4 per cent with an operative mortality of 2.9 per cent.

Our own smaller series of 24 cases indicates the same experience in our clinic, for as time has gone on and a more complete understanding of the life history of these tumors has been made available, fewer recurrences and second operations take place.

Cerebral Astrocytoma.—Whereas the cerebellar astrocytoma is characteristically cystic, the same tumor in the cerebrum may or may not be cystic. If present, the cyst is usually multilocular and has many small, connecting cavities with masses of soft, degenerate, almost gelatinous tissue separating them. We have found cerebral astrocytomas only in adult patients. They tend to recur at a far earlier date and have generally a much less favorable prognosis than those tumors of the cerebellum with a similar cytological nature.

These tumors usually lie beneath the cortex, sometimes at a considerable depth, though not infrequently they reach the surface as purplish-gray masses. They may attain an enormous size and very frequently are found in the temporal or frontal lobes where they produce clinical symptoms which make them reasonably easy to recognize. Consequently, they should be operated upon early when symptoms first appear and operative results may be expected to be more favorable.

It is our custom to attack these tumors in a radical manner and the development of the electrosurgical unit has made this a far more satisfactory and practical procedure than has been true in the past. Often one is surprised to see what appears to be a very definite line of demarcation between these tumors and the surrounding normal brain tissue, almost as if they were encapsulated. As the resection is continued more deeply, however, this line of division becomes less definite and one finds infiltrating masses of tumor tissue spreading root-like in many directions. In the softer, more cystic and degenerated areas one may sometimes rapidly remove large amounts of tumor with the suction apparatus. It has been suggested that surgical attempts to remove these tumors radically causes them to become more malignant, but this has not been borne out in our clinic by careful microscopic studies of the tumors removed at the second, or even the third operation. Certainly, every intracranial tumor which can be definitely localized should be attacked surgically with the thought in mind of removing it as completely as possible. When the patient's history and physical findings indicate the possible presence of an intracranial tumor every diagnostic step must be taken to confirm or deny its existence. To pneumoencephalography, ventriculography, angiography, electroencephalography, may now be added the use of the radioactive isotope tracer dye test. All of these laboratory tests are valuable corroborative evidence of the results of an indispensable carefully performed neurological examination.

The following case summary illustrates a procedure deemed necessary in the past which at present is unnecessary because of refinements and additions to diagnostic methods.

A young man complained of momentary seizures of faintness, preceded by a bad odor and taste. These became more frequent, but were never followed by a localized motor convulsion or a generalized epileptic attack. Slight headaches occurred frequently, but there were no other symptoms though repeated physical examinations were made. Within five days, the patient developed a bilateral papilledema with retinal hemorrhages and it was obvious that an intracranial space-occupying lesion was present.

A ventriculogram was performed and although the ventricular system was well-filled, no deformity could be found after a careful study by the entire staff. Within the next year, the patient had returned to work after a right subtemporal decompression had been performed. His papilledema subsided completely but his uncinate seizures continued. Finally, a right facial weakness became apparent and another ventriculogram was made which demonstrated a deformity of the tip of the inferior horn of the left ventricle. An osteoplastic craniotomy was performed and the anterior pole of the temporal lobe, which contained the tumor, was removed.

In our series there have been 167 astrocytomas of the cerebrum and 24 of the cerebellum. Of these 191 patients, 33 have lived over five years; 28 patients have lived from two to five years; 8 from eighteen months to two years; 20 from twelve to eighteen months; 15 from six to twelve months; 23 from zero to six months; 30 were deaths in the hospital at varying periods following operation and 3 patients died in the hospital after admission without operation. Thirty-one patients have failed to respond to our latest follow-up letters and of these, 2 were alive over five years; 11 were alive from two to five years after operation; 3 from eighteen months to two years; 2 from twelve to eighteen months; 4 from six to twelve months and 9 from zero to six months.

The astrocytomas are classified microscopically according to two cell types: *fibrillary* and *protoplasmic*. It probably never occurs, however, that a tumor consists entirely of fibrillary astrocytes to the exclusion of the protoplasmic and *vice versa*. The fibrillary astrocytoma may be a solid tumor and hyalinized, even calcified, areas may be found but it is more common for these tumors to be cystic, probably because they are poorly vascularized. The nuclei stain darkly, are widely spaced, are of a fairly uniform oval to round shape and are surrounded by but a scant cytoplasm. Amitoses are commonly seen, whereas mitoses are rare. With the aid of such stains as Cajal's gold-sublimate or phosphotungistic acid hematoxylin, one may see a rich, felt-like ground work of interlacing neuroglial fibrils in which the nuclei appear to be imbedded. These fibrils are the processes which rise from and characterize the adult fibrillary astrocyte. There is the occasional tumor which must be accepted as an astrocytoma but yet which shows occasional mitoses, a variation in nuclear sizes and shapes, areas of suggested palisade formation and cells, some of which are frankly astroblasts. Such a

FIG 50 — Photomicrographs of astrocytoma fibrillare: (*A*) low power, × 75; (*B*) high power, showing the pronounced fibrillary background, × 350.

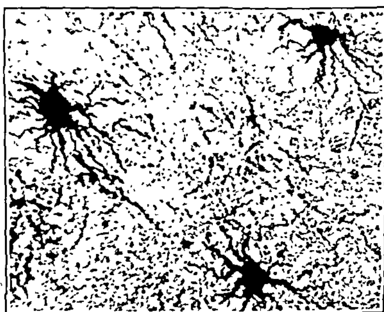


FIG 51 — Photomicrograph of astrocytoma fibrillare stained by Cajal's method, showing the fibrillary processes of the adult astrocyte, × 570.

tumor is obviously more malignant than the usual fibrillary astrocytoma, and it serves to demonstrate first, the fact that tumors merge from one class into another of a different malignancy and second, that they cannot always arbitrarily be placed into clean-cut categories. (Figs. 50 and 51.)



FIG 52 —Photomicrographs of astrocytoma protoplasmaticum. (*A*) low power, × 100, (*B*) high power, showing the meaty character of the cells without fibrillary processes, × 500.

Protoplasmic astrocytomas, less common than the fibrillary type, frequently contain many astroblasts. These tumors, grossly much like any other astrocytoma, are made up of adult astrocytes which contain darkly-staining nuclei eccentrically placed in a fairly generous cytoplasm. The fibrils are blunt, meaty and less readily stained

than those of the fibrillary astrocytes. The low power field shows the protoplasmic astrocytoma to be less cellular and the striking meshwork of fine fibrils is missing. These tumors are also relatively avascular, show areas of degeneration and contain few mitotic figures. (Fig. 52.)

Astroblastoma.—These interesting tumors should be thought of together with the astrocytomas. The *astroblastoma* is a more malignant tumor, however, because it is formed of a more primitive, more rapidly growing cell, the astroblast. Its relative malignancy lies between that of the glioblastoma and the astrocytoma and surgically, they may be mistaken for either one of these tumors. Clinically, they afford no symptoms by which they might be differentiated from any of the other more common tumors arising from the glial elements. To justify the diagnosis of astroblastoma certain characteristic criteria must be found and very frequently these criteria are to be found only after repeated, careful studies of sections taken from several different parts of the tumor stained by methods which will reveal the tell-tale features of the tumor. Cushing has recorded only 35 of these neoplasms in his series of 2,000 verified intracranial tumors and we have definitely diagnosed but 6 such tumors in our own series. No doubt a careful combing through of the glioblastoma material would reveal several additional astroblastomas. A striking fact is that in 31 of his cases of astroblastoma Cushing performed 58 operations, which gives some idea of their survival period. There were 10 fatalities which gave a case mortality of 32.2 per cent; this may be an indication of their increased malignancy and frequent large size when first seen at operation.

The astroblastomas occur principally in the cerebral hemispheres. They may be partially cystic with areas of solid tissue which is quite dense and they often attain a very large size before they can be localized by ordinary clinical means. Like certain others of the glioma group, they sometimes are fairly well demarcated and appear almost enucleable. Microscopically, they are one of the most interesting of all intracranial tumors and their structure as seen under the low power is characteristic and unmistakable. Many blood-vessels may be present, the intima of which (not the muscularis) is hypertrophied and frequently of a hyalin appearance. The cells are numerous but they give the appearance of having been shaken loosely into place. They may be lined up almost in columnar order along certain of the vascular sinuses, and in such a field the identifying feature, the "sucker foot," may be found and studied in detail with the oil lens. The cells are predominantly triangular in shape, with one or two blunt processes and a long one which may reach a vessel and appear to attach itself to the vessel wall by a simple

bifurcation, "sucker," mechanism. The astroblast should never be confused with a cortical ganglion cell because of its triangular shape. It is a smaller cell; it never contains tigroid material; it may contain one to three nuclei and its fibrils are stained by the same stains used for any neuroglial fibrillæ. (Fig. 53.)



FIG. 53.—Photomicrographs of an astroblastoma: (*A*) low power, showing the uniform and triangular shape of the cell with palisading along the hyalinized blood-vessels, × 210; (*B*) high power, showing the attachment of sucker feet to a hyalinized blood-vessel, × 1080. Phosphotungstic-acid hematoxylin stain.

The 6 astroblastomas in our own series died in the hospital following operation.

Oligodendroglioma.—Among the more slow-growing, less malignant of the gliomas are the *oligodendrogliomas*, the clinical course of

which averages from five to six years. They constitute approximately 4 per cent of the glioma group. They are solid tumors which, in our experience, have been found only in the cerebrum of adults but they have been reported found in the cerebellum as well, and even in the brains of children.

Grossly, these tumors are very firm, reddish-gray in color and like some of the other less malignant gliomas may have an appearance of demarcation, even of encapsulation. During the course of their slow growth, during which time they may assume tremendous proportions, they commonly become calcified, so that they may often be localized by areas of spotty, flaky calcification which appear in the roentgen-ray films and lead to the preoperative diagnosis of oliodendroglioma.[1]

The story of a sixty-one year old executive illustrates many of the characteristics of these tumors. For three years, his secretary had noted that his vision to the right of the midline was defective and that he was having more and more difficulty in getting the day's work done to his own satisfaction. It was not until six months before he entered the hospital that he complained of tiredness, difficulty in expressing himself, clumsiness in the use of his right hand and remarked to his wife that he seemed to bump into objects to the right of him as he walked.

This man had a well-defined right homonymous hemianopsia, a verbal aphasia, a right hemiparesis, and loss of stereognostic sense in his right hand. At no time had he ever complained of headaches and there was no papilledema.

Roentgen-ray examination of the skull showed scattered small areas of calcification in the left parieto-occipital area and upon operation a subcortical oligodendroglioma was removed from the parietal lobe. The tumor was yellowish-red in color and fairly well demarcated from the surrounding brain tissue. His postoperative recovery was uneventful and his hemiparesis and aphasia improved so markedly that he was able to return to his work. He continued well for three years, but succumbed after a prostatectomy operation.

The long course of this patient's illness and the presence of the calcifications visualized on the roentgen-ray films led us to make a preoperative diagnosis of oligodendroglioma. In the absence of signs and symptoms of increased intracranial pressure, one must be careful not to fall into the error of diagnosing such cases as cerebral vascular accidents.

A man aged thirty-eight years complained of headache, vertigo, and frequent vomiting for eight months, and on several occasions had fallen to the right. He was lethargic, disinterested in his surroundings, unable to concentrate on a subject of conversation and was unable to give a connected story of his illness. He had a swelling of the left disc, but none of the right, and there was some facial weakness on the left side. He was left handed.

[1] The oligodendroglia cell has different functions. They are found about the larger ganglion cells of the cortex and basal ganglia and also along myelinated nerve fibers in the white matter. It has been suggested that they regulate the formation of myelin and act as intermediary agents in the exchange of metabolic products between ganglion cells and brain fluids. Under certain acute toxic conditions they act as a type of scavenger cell.

Roentgen ray films showed spotty areas of calcification in the left frontal area and large meningeal artery channels in the bone. Preoperatively he was believed to have a meningioma or a calcified glioma of the left frontal lobe.

A left transfrontal osteoplastic craniotomy revealed a solid, partially calcified, rose-gray tumor which contained a large cyst full of a thick chocolate-colored fluid. Because of the extent of the tumor and the extreme vascularity of the skull, actual removal of the tumor was accomplished by a second stage operation. At that time the anterior two-thirds of the left frontal lobe was



FIG. 54.—(*A*) Calcification in an oligodendroglioma of the left temporal lobe; (*B*) photomicrograph of an oligodendroglioma showing the clear cytoplasm, definite cell boundaries, and round centrally placed nuclei typical of this tumor. There is an abundance of calcium deposits in this particular tumor, × 635; (*C*) same tumor at a magnification of 1680.

removed (85 gm.), with a complete removal of all tumor tissue. Microscopically, the tumor proved to be an oligodendroglioma, rich in calcium and ontaining many necrotic areas

The patient made a good recovery, became much brighter and more alert mentally, had no postoperative weakness of his extremities and returned to work at his home in another city.

Ordinarily, the oligodendrogliomas have a characteristic appearance, though a careful search may be required to find a typical microscopic field. The cells are numerous, the nuclei are round, stain deeply and are of uniform size. A pale staining halo of cytoplasm surrounds each nucleus. Mitotic figures are rarely found. When special neuroglial fibrillæ stains are used, a few short radiating processes may be seen interlacing in the intercellular substance but these processes are notoriously difficult to stain. With ordinary hematoxylin-eosin the fibrils do not stain and the cells appear to be fitted together in a mosaic-like fashion. These are not ordinarily vascular tumors and, in keeping with their slow growth, necrotic areas and flecks of calcium are frequently found. One may also find foci of fatty degeneration of the cells, where the cells are large, swollen and vacuolated. Some fields may contain what appears to be adult fibrillary astrocytes. (Fig. 54.)

We have had 30 oligodendrogliomas in our series of verified intracranial tumors. Of these we have been able to follow 26 patients of whom one patient is living twenty-five years following operation. Five patients are alive after five years, 7 lived from two to five years; 3 lived from eighteen months to two years; 1 lived from twelve to eighteen months, 1 lived from six to twelve months; 2 lived from the time of operation to six months and 6 patients died in the hospital following operation. Of the 4 patients we have been unable to contact, 1 was followed from two to five years; 1 from eighteen months to two years, 1 from twelve to eighteen months and 1 from six to twelve months.

Polar Spongioblastoma.—These tumors comprise approximately 2 per cent of all the gliomas and, unfortunately, they commonly occur in surgically inaccessible portions of the nervous system. Otherwise the fact that they are slow-growing and relatively benign would make them more amenable to surgical interference Such tumors are frequently found in the optic chiasm or nerves of children, where they are known as chiasmal or optic gliomas. As was true in some of our patients, they are quite likely to be accompanied by peripheral manifestations of neurofibromatosis, or von Recklinghausen's disease. They may produce an erosion of the anterior clinoid processes and optic foramen which is easily demonstrable in the roentgen-ray films *Polar spongioblastomas* frequently occur on the undersurface

of the cerebral hemispheres, where we have found them destroying the cerebral olfactory apparatus and basilar levels of the thalamus. They not infrequently occur in the pons and midbrain of children and young adults, where they are completely inaccessible and uniformly fatal.

Surgical experiences with the chiasmal spongioblastomas are discouraging, inasmuch as the tumor frequently involves the midbrain as well as the chiasm. In our patients, symptoms of involvement of the hypothalamus were prominent features of the clinical picture. If such a tumor could be exposed when it was small and localized to one optic nerve, or to the chiasm alone, complete removal might be possible, though blindness would be the result.

Grossly, the optic chiasm and optic nerves, when destroyed by such a tumor, are hard, dull red in color, and enormously enlarged. When present in the cerebrum proper, the tumor may be either very necrotic and cystic, or it may be quite firm, white and avascular. Due to the difficult location in which they are usually found, a complete removal is not always possible.

Tumors completely composed of unipolar or bipolar spongioblasts are rare and in those tumors specifically called polar spongioblastomas, the name is applied because the polar spongioblast is the predominating cell. Other less readily recognized neural and glial elements are also present and in turn, many polar spongioblasts may be found in other tumors, such as the glioblastomas. Polar spongioblasts, while young cells, are more differentiated than are the primitive, non-polar spongioblasts which are genetically close to the primitive medullary epithelium and which comprise other rarer and more malignant gliomas.

The typical polar spongioblastoma may, at first view under low power magnification, suggest an acoustic neurinoma or a neurofibroma. Large coils and whorls of parallel streaming fibrils with a moderately dense field of oval nuclei are seen when the section is cut longitudinal to the stream of the fibrils. When the section passes at right angles to the stream, the nuclei appear small, round and dark and the long graceful sweep of the processes is not seen. Some cells have but one process; others have two. These processes are coarse and hard in appearance as compared to the delicate wavy fibrils of an acoustic neurinoma. Being essentially indolent tumors, mitoses are rare and there may be central areas of necrosis. A common tendency is for the bundles of cells to arrange themselves in rows and in solid formations, particularly near small vessels. (Fig. 55.)

One patient, an adult, from whom a section of a glioma of the chiasm was made, was given two series of deep roentgen-ray therapy

and remained alive for twenty-six months. Because of the inaccessibility of the tumor, other patients have had even a less favorable outcome.

A man, aged sixty-eight years, was said to have had a "stroke" eighteen months before his first neurological examination and a reliable informant stated that the patient had had a coronary thrombosis several months before admission to the hospital This was confirmed by electrocardiographic studies at the time he was being studied neurologically. He had complained of smelling bad odors at frequent intervals; he became careless in his appearance and obscene in his speech, he slept a great deal and showed some weakness of the right side of the body. It was difficult to differentiate between a left-sided vascular lesion or a glioma of the left side, and not until after ventriculography was done was it obvious that he had a large space-occupying lesion deep within the left frontal and temporal lobes.

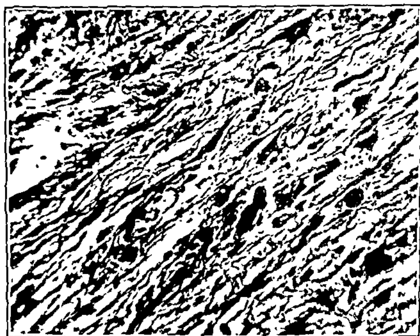


Fig 55 —Photo micrograph of a spongioblastoma unipolare stained by Cajal's method, × 350

Operation was performed under a local anesthetic, but the tumor was found to be surgically irremovable He did well following a generous decompression, but ten days after operation he suffered a second coronary thrombosis, rapidly developed pneumonia and died Autopsy revealed a large, white, firm tumor occupying a site deep within the fronto-temporal region, destroying much of the basilar ganglia on that side and invading the pons and midbrain It was *a spongioblastoma with an unusually high percentage of bipolar spongioblasts* with large, thick, unwaving processes

There are 10 patients in our series with polar spongioblastoma. Eight were followed and 1 patient is still alive seventeen years after operation, 1 lived over five years; 1 lived two to five years; 1 lived

eighteen months to two years and 4 patients died in hospital following operation. We have been unable to obtain a follow-up record on the 2 remaining patients after they were discharged from the hospital following operation. In Cushing's report, the survival period from the onset of symptoms in this group of patients was forty-six months and his case mortality and operative mortality were 25.8 and 22.2 per cent, respectively. We have not found that roentgen-ray or radium therapy uniformly has a beneficial effect, though it may be a useful adjunct in certain of the optic gliomas.

Medulloblastoma.—The *medulloblastomas* constitute 6 per cent of all the gliomas in our series and occur most frequently in the cerebellum of children.[1] These tumors show a predilection for the structurally complicated region of the roof plate of the fourth ventricle. Clinically, they have been grouped under the common term of "midline cerebellar" tumors.

It will be remembered that in the early histogenesis of the central nervous system, the medullary plate consists of a single layer of columnar epithelial cells, the medullary epithelium. Coincidental with the thickening of the neural plate, small round vesicular cells undergoing mitosis appear among these columnar cells. They are known as germinal cells, some of which differentiate into neuroblasts, while others continue to give rise to indifferent cells, known as medulloblasts, the future development of which is unpredictable. While the potentiality of these cells cannot be determined, there is no doubt that they are migratory, are present in large numbers, remain fixed in an undifferentiated stage of development, and give rise to large tumor masses.

The most common and striking symptom of a medulloblastoma of the fourth ventricle is recurrent and persistent attacks of vomiting of the projectile type occurring most frequently with changes in posture. Repeated vomiting spells when the child arises in the morning gradually cease as the intracranial mechanics become adjusted to the upright position. The vomiting is due to stimulation by the tumor of the vagus centers in the medulla, but it is not difficult to understand how such periodic attacks of vomiting may be ascribed to gastro-intestinal disturbances, or to other causes far removed from the central nervous system. The unfortunate circumstance in the history of these patients is that such diagnoses are often persisted in to the point where repeated roentgen-ray examinations of the intestinal tract and needless surgical measures are performed.

Alternating with vomiting spells, attacks of severe, excruciating headache constitute the second most important early symptom. The headaches are invariably suboccipital and always are accompanied

[1] Cushing has described 18 tumors of the cerebral hemispheres which have been subsequently verified as medulloblastomas, but we have not as yet encountered such a tumor in the cerebrum. Contrary to the cerebellar medulloblastomas they are relatively benign and their life history resembles the oligodendrogliomas.

by suboccipital pain and tenderness upon palpation or percussion. This leads to a fixed position of the head in which the occiput is tilted toward one shoulder or the other as the result of the patient's attempt to relieve his pain. Flexor movements of the neck are resisted and are accompanied by an increase in the patient's discomfort. It may be understood how a tumor lying above the roof of the fourth ventricle will quickly occlude the cerebrospinal fluid pathway. Such an occlusion produces an obstructive secondary dilatation of the ventricular system cephalad to the lesion. The resultant hydrocephalus is rapid, the rise in intracranial pressure great, and the accompanying headaches unbearable. (Fig. 56.)



FIG. 56.—Series of photographs of a patient with a midline cerebellar tumor: (A) three weeks after operation at age of three years, (B) after deep roentgen-ray therapy at age of six years, (C) after second course of deep roentgen-ray therapy at age of nine years, (D) after second operation at age of thirteen years.

So it is that the yielding suture lines of a child's skull become separated; the cranial bones become thinned out from convolutional pressure atrophy; the optic nerves become swollen and edematous; and visual acuity becomes quickly impaired. These mechanical factors affecting the cranium produce a tympanitic note upon percussion of the skull, a sign first described by Macewen.

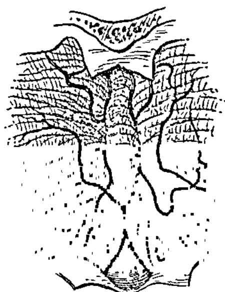


FIG 57.—Operative sketch showing the nodule of medulloblastoma appearing between the cerebellar tonsils and extending into the spinal canal.

Contrary to what one might expect, spontaneous or induced nystagmus is not a common finding in the examination of these patients. If a horizontal or vertical ataxic movement of the eyeballs is present upon fixation, it is clear that involvement of the cerebellar hemispheres or of the vestibular connections with the oculomotor nerve nuclei in the brain stem has occurred. Cerebellar symptoms other than a trunkal ataxia are not present commonly in cases of medulloblastoma, although varying degrees of dysmetria may occur. A definite hypotonia of the musculature is quite characteristic and walking is performed with a wide base and the gait is uncertain with a tendency to fall backward.

Advantage may be taken of dogmatism to emphasize this not uncommon clinical syndrome in children more briefly. One should suspect a midline cerebellar tumor in a child with a syndrome of morning vomiting, suboccipital headaches, an enlarging head, choked discs, and a cerebellar type of ataxia.

At operation the medulloblastomas may or may not be visible on the surface, although very frequently a portion of the tumor may be

seen to project into the fourth ventricle, or even into the spinal canal, between the cerebellar tonsils. In other cases, the vermis of the cerebellum appears wide and prominent and by splitting this structure with the electrosurgical unit, the purple, soft, vascular tumor mass extrudes into the field. (Fig. 57.) Not infrequently one or both cerebellar hemispheres are invaded by the tumor and in one such case, the tumor tissue was spread over the surface of both cerebellar lobes. A sagittal section of an autopsy specimen shows very well the enormous extent of these tumors along the brain stem. (Fig. 58.)

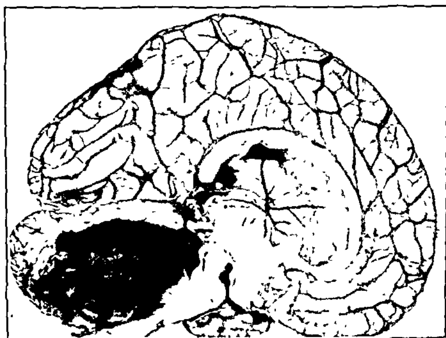


FIG 58.—Sagittal section of autopsy specimen which shows the origin and extent of a medulloblastoma as well as an enormous secondary hydrocephalus.

There is a risk, and probably a certainty, that radical surgical attempts at removal may be followed by an inoculation of the cerebrospinal fluid spaces and this has occurred in three of our patients, all of whom later developed a transverse lesion of the spinal cord due to tumor tissue which had grown rapidly in the spinal subarachnoid space, after being "seeded" there from the original tumor within the cranium On the other hand, it is the most certain and immediate way of obtaining symptomatic relief Postoperative radiation should be started as soon as possible and we have made it a practice to distribute the radiation over the entire cerebrospinal axis Because of their primitive cell type, these tumors are among the most susceptible of all intracranial tumors to the effects of deep roentgen-ray or radium therapy.

In our series of 37 cases of medulloblastoma, the ages of the patients have ranged from one to twenty-six years. These tumors are highly malignant, as might be judged from their low developmental position; they grow rapidly and, as stated above, tend to inoculate the cerebrospinal fluid and spread widely in its spaces.

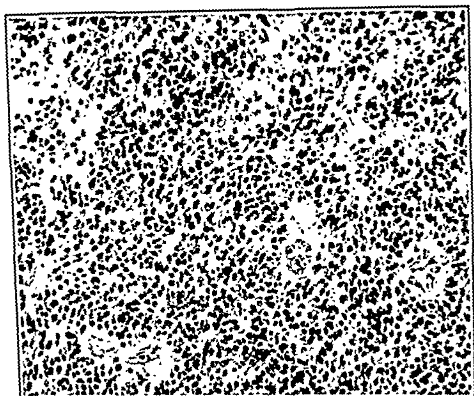


FIG 59.—Photomicrograph of a medulloblastoma showing the uniformity of the hyperchromatic nuclei and their tendency of arrangement into small groupings by a scanty connective tissue stroma, × 220

To the inexperienced, a medulloblastoma may appear both grossly and microscopically like a so-called meningeal sarcomatosis, but meticulous study of the cells, as stained by selective methods, will readily reveal the characteristics of a medulloblastoma. This is a highly cellular tumor and the tissue is usually densely packed with small round nuclei which stain intensely and which contain many mitotic figures. These cells are, in the main, young medulloblasts but uni- or bipolar spongioblasts; large, clear, oval neuroblasts and even astrocytes may be found in varying quantities. A frequent formation of the cells is that of a perivascular pseudo-rosette and in places, great masses of cells are packed into clusters, in the center of which there may be a clearing. The tumor is frequently very vascular with many small vessels threading their way everywhere and between them pass a fine network of delicate cytoplasmic

streamers from the medulloblasts and neuroglial fibrils from the astrocytes. Connective tissue is extremely sparse. We have had an opportunity to study one of these tumors following deep roentgen-ray therapy. There was a reduction in the number and size of the nuclei, which appeared generally pyknotic, and there was a marked proliferation of collagenous connective tissue which enclosed a few small islands of viable tumor cells. Histologically analogous but of rarer occurrence are the retinoblastomas and sympathicoblastomas. (Fig. 59.)

Cushing reported 99 operations upon 64 patients with medulloblastomas with 25 postoperative deaths, thus giving a case mortality of 39 per cent and an operative mortality of 25.2 per cent.

Of the 37 patients in our series, 2 lived over five years; 4 lived from two to five years; 2 lived from twelve to eighteen months; 4 lived from six to twelve months; 4 lived from the time of hospital discharge to six months and 16 patients died in the hospital. Two patients entered the hospital and died without an operation but were verified at autopsy examination. Three patients could not be followed after intervals of six, twelve and eighteen months.

Ependymoma.—Like medulloblastomas, the *ependymomas* show a predilection for the roof plate of the fourth ventricle, though we have found them deep within the cerebral hemispheres. When they lie in the posterior cranial fossa any place below the tentorium cerebelli, they frequently produce the syndrome of a "midline cerebellar tumor" and in so doing, may appear clinically very much like a medulloblastoma They are not particularly common and our 24 cases constitute but 1.8 per cent of all our verified intracranial tumors.

The clinical story of these tumors is well illustrated in the two following résumés:

The first symptom noted in a thirteen-year-old boy was vomiting, which occurred persistently each morning and began in June of 1927. He was hospitalized and placed on various diets in an effort to correct what was diagnosed as a duodenal obstruction. Vertigo and diplopia had been complained of infrequently since the preceding April, but became pronounced in July. Suboccipital headaches began in August and gradually became worse He then complained of pain and tenderness in his neck.

The boy's head was held with the occiput tilted decidedly to the right. There was marked tenderness on percussion and palpation over the suboccipital areas Marked hypotonia was present, but movements of precision were well performed with the arms and legs. A bilateral papilledema was so marked that visual acuity was diminished. The left external rectus muscle was paretic. A coarse nystagmus was present on looking to the right. Roentgen-ray films of the skull showed prominent convolutional markings and separation of the suture lines

tion of a tumor mass was found between the cerebellar tonsils. As these were separated a large, firm mass was found lying upon the floor of the fourth ventricle. All attempts to do more than remove the lower prolongation of the tumor were followed by alarming vasomotor and respiratory responses.

The boy made an excellent recovery and gained weight. He was given intensive deep roentgen-ray therapy, but succumbed twenty months later with symptoms of an acute obstruction to the flow of cerebrospinal fluid.

Sudden, severe attacks of vomiting, with no other complaints, began in a girl of eighteen years who had always been in the best of physical condition. Eventually, eight months later, she developed a left-sided weakness of the face and arm, headaches, a high grade papilledema and a left homonymous hemianopsia. She was believed to have a right temporal lobe tumor and accordingly was operated upon with the removal of a large, fairly soft and vascular tumor deep within the right temporal lobe. No connection between the tumor and the ventricle could be found. She made an excellent recovery, but her headaches returned after a year and she was given a course of deep roentgen-ray therapy. Her complaints disappeared for over another year, at which time she again complained of headache and some vomiting. Again deep roentgen-ray therapy was given with little benefit. Four years after operation, she succumbed

The tumor was an ependymoma of somewhat unusual architecture and contained cells which showed occasional mitotic figures. Although as complete a removal was done as could be judged by gross appearance, it is obvious that there was a recurrence which at first was controlled by deep roentgen-ray therapy. It is reasonable to believe that this particular ependymoma, made up of young cells and containing mitotic figures, should show some susceptibility to such treatment.

Grossly, the ependymomas are frequently firm and appear to be enucleable. So much is this true that there is always a strong temptation to attempt to total removal; but due to their dangerous position such attempts may produce alarming symptoms, if not a fatality. When they lie in the fourth ventricle, they project downward between the cerebellar tonsils, through the foramen magnum, and into the spinal canal far more regularly than do the medulloblastomas and in this position, they may be dangerously caught in branches of the posterior inferior cerebellar artery, or they may exert mechanical pressure upon the floor of the fourth ventricle. In such a location, it is frequently impossible to remove the entire tumor and it may be necessary to remove the laminæ of the atlas and the axis, in order to provide an adequate decompression. One patient in this group, a young woman of twenty-three years died in her bed of sudden respiratory failure. Upon examining the brain at autopsy there was discovered an enormous ependymoma growing upward from the anterior extent of the fourth ventricle, through the substance of the cerebellum, out upon the upper surfaces of the cerebellar hemisphere like a giant, tough, gray-colored oyster. (Fig. 60.)

Microscopically, two types of cells are found in these tumors; adult ependymal cells and ependymal spongioblasts, or "ependymoblasts." In our experience, tumors made up predominantly of ependymal spongioblasts, the so-called ependymoblastomas, are rare as compared to the ependymoma or adult cell type of tumor. The cells of the ependymoma have a moderately large, clear nucleus and a fair amount of cytoplasm. Cell processes are lacking but the cytoplasm may be stained to show blepharoplasten, which are dust-

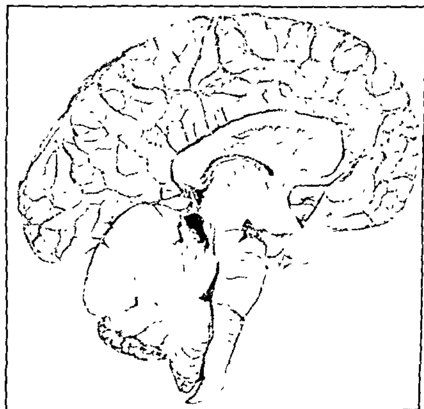


FIG. 60 —Mid-sagittal section of brain showing an ependymoma arising from the region of the anterior medullary velum.

like inclusion bodies characteristic of ependymal cells. There are no mitoses. There is usually no specific architectural arrangement to the cells, though they do tend to arrange themselves in ropes or masses and sometimes, as in the case of cerebral ependymoma, the perivascular, ring-like arrangement of the cells is very striking. True rosettes are rare but pseudo-rosettes are a common finding. A scanty stroma may be demonstrated by special stains. The ependymoblastomas, like the ependymomas, are very cellular tumors, the cells of which not only show blepharoplasten, but also have tail-like processes which may be quite long and mitotic figures are occasionally found. (Fig. 61.)

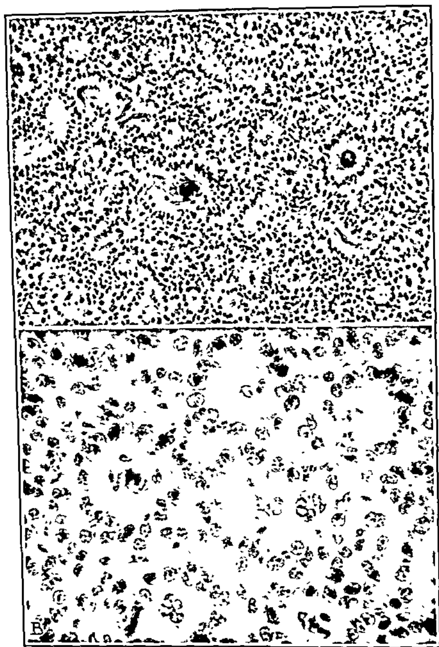


FIG. 61.—Photomicrographs of an ependymoma: (*A*) low power, showing pseudo-rosette formation of the cells about blood-vessels, × 190; (*B*) high power, × 520.

Ependymal gliomas are not considered to be amenable to deep roentgen-ray therapy, at least not to the extent that certain other tumors are, such as the medulloblastomas. But that repeated courses of such postoperative treatment, where an adequate decompression opening has been made at the time of operation, may be of unquestionable value is demonstrated by the history of the patient with cerebral ependymoma.

Of our 24 patients with ependymomas, 1 is alive over ten years; 2 have lived from two to five years; 1 from eighteen months to two years; 1 from twelve to eighteen months; 2 from six to twelve months; 3 from the time of hospital discharge to six months, and 9 died in the hospital following operation. One patient died in the hospital without having been operated upon and we have been unable to contact 4 patients at this time. Of these, 1 patient was followed from twelve to eighteen months and 3 could not be reached after discharge from the hospital.

Papilloma of the Choroid Plexus.—Cystic degeneration of the choroid plexus and calcified masses within the plexus are common findings in many normal brains but true tumors of the choroid plexus are among the rarer of intracranial tumors. Cushing and Davis[1] reported upon 6 of these tumors which occurred in 1,000 verified intracranial tumors, and since that time 6 others occurred in Cushing's succeeding 1,000 cases. Of these 12 papillomas, 6 were midline tumors of the fourth ventricle; 3 were in the cerebello-pontile angle, and 3 were associated with large cysts of the lateral ventricles.

These tumors, arising originally from the medullary epithelium, are true tumors of the brain and should be considered with the gliomas. They are completely benign and may grow to an enormous size before definite clinical symptoms result We have seen such a tumor, as large as a small head of cauliflower and of very much the same gross appearance, arising from the foramen of Monro in a sixteen-year old girl. It had produced a tremendous hydrocephalus and surgical access to it was very easy through the enormously dilated lateral ventricle into which it had grown and which it had completely filled. It was removed down to its stump-like origin at the foramen, where it had greatly distorted the walls and floor of the third ventricle. Now, five years later, the girl's only symptoms remain as before operation; that is, *a left hemiparesis and amenorrhea.* That the tumor has regrown to a considerable size is shown by the slowly increasing fullness and tightness of her decompression opening.

[1] Cushing, H., and Davis, L. Papillomas of the Choroid Plexus, Arch. Neurol and Psychiat., 13, 681, 1925.

The microscopic picture of *choroidal papillomas* is a simple one. High, vascular tufts with cores of connective tissue are covered by columnar cells containing mitochondria, but no blepharoplasten. Except for this absence of blepharoplasten, the individual cells resemble those seen in the ependymomas. (Fig. 62.)



FIG. 62.—Ph[illegible] (*A*) low power, showing the [illegible] nar cells which cap a central [illegible] owing details of columnar cells, × 345.

Pinealoma.—Tumors arising in the pineal gland are among the rarer gliomas. Because of the anatomical position of the pineal gland, its enlargement by a neoplastic growth causes early blockage of the aqueduct of Sylvius and a resultant internal hydrocephalus, damage to the vegetative centers of the hypothalamus by the extreme thinning of the walls and floor of the third ventricle, and invasion of the corpora quadrigemina and other important nuclear centers of the midbrain. Clinically, such patients show a paralysis of conjugate

upward movements of the eyeballs and there is usually a rapid increase in the severity of their headache, nausea, vomiting and failing visual acuity. It is a moot point as to whether or not the pineal gland plays some rôle in the endocrine physiology of the body, but there is both experimental and clinical evidence to lend support to such a proposition, especially in the young individual.[1] The syndrome of "pubertas precox" has frequently been linked to pineal gland pathology.

Surgery of the pineal gland is singularly disappointing in most cases. To remove the tumor, usually through a transventricular approach, and not injure the great cerebral veins and vital parts of the midbrain, is a tremendous task. These tumors are frequently very tough, adherent to the surrounding structures, and so calcified as to be diagnosed accurately from the roentgen-ray pictures alone. They may "seed" themselves by means of broken-off cell masses throughout the intracerebral ventricular system. In a series of 14 verified pineal gland tumors, 6 of which were operated upon, Cushing had an operative mortality of 75 per cent, and a case mortality of 100 per cent. In later years, probably through improvement in operative approach, several successful removals have been reported. In Cushing's series, 5 of the patients were boys twelve years or younger, and 3 were girls less than fifteen years old. In most of the preadolescent and adolescent children, some aberration of sexual and somatic development was prominent. The adult patients complained of the symptoms of rapidly increasing intracranial tension, incoördination of the eyes and certain symptoms referable to the hypothalamus. We have operated upon five such patients; one a girl of eighteen years whose tumor was a large, mossy mass which had destroyed most of the brain between the mid-portion of the third ventricle and the pons and which was filled with fine flecks of calcium. Another patient was a middle-aged man whose enormous tumor extended all the way back through the fourth ventricle into the right cerebellar hemisphere and who died a sudden respiratory death on the table while the scalp incision was being made.

The pineal anlage is an outgrowth of the true brain and the more primitive and malignant type of pineal tumor, the *pineoblastoma*, is made up of densely packed pineal parenchymal cells with small round nuclei which stain darkly, all of which have very much the same appearance and which may be richly supplied by many fine vessels. The *pinealoma* shows considerably more differentiation. It is made up of two types of cells, mingled together in places, but frequently found in nests or "islands," almost free of any cells of

[1] Davis, L., and Martin, J. Results of Experimental Removal of Pineal Gland in Young Mammals, Arch. Neurol., and Psychiat., **43**, 23, 1940

the other type. One cell is a small, dark, lymphocytic type of cell, no processes of which are ever seen. The other cell has a large, clear, vesicular nucleus and a generous cytoplasm which contains blepharoplasten. With special stains, some of these large cells may be shown to have short thick processes ending in a blunt dilatation. Pinealomas are commonly bloody tumors and because of their calcium content may be actually gritty to palpation. (Figs. 63 and 64.)

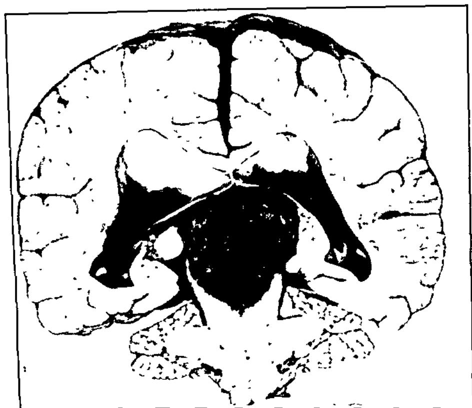


Fig. 63.—Coronal section of brain showing the gross appearance of a pinealoma.

Ganglioneuroma.—The vagaries of the occurrence of tumors in any particular series is exemplified by the fact that in our own series we have encountered 2 *ganglioneuromas*, although Cushing, in his report of 2,000 verified tumors, records only 3, and he, therefore, regards them as pathological curiosities which hardly deserve surgical consideration.

In our own series, convulsive seizures over a period of two years characterized the clinical story in a woman of middle age. Localization was impossible by neurological examination alone but calcification was seen within the left cerebral hemisphere upon the roentgen study of the skull. Because epilepsy had developed suddenly in an adult, an encephalographic study was made. It was found that the ventricles were pushed to the right and the left ventricle was de-

formed. At operation a firm, hard mass was found in the left frontal lobe. The other patient, a veteran of the War of 1914–18, suffered paroxysmal pains in the right side of the face throughout the area of distribution of the maxillary and ophthalmic divisions of the



Fig. 64 Photomicrographs of (A) pinealoma showing large pineal parenchymal cells and the predominating lymphoid cells with hyperchromatic nuclei, × 630. (B) pineoblastoma showing the primitive pineal anlage cell and calcareous deposits, × 340

trigeminal nerve He had received several alcohol injections, some with partial relief, but meanwhile he developed other neurological symptoms such as bilateral papilledema with some atrophy of the nerve heads, headaches and general weakness He did not have a trigger zone It was believed that he did not have a typical major

trigeminal neuralgia but that an intracranial neoplasm was the cause of his intractable pain. A subtemporal approach was made to the Gasserian ganglion. Above it, attached to the undersurface of the brain, was a pea-sized, sessile nodule of firm, yellow tumor tissue which pressed upon but was not intimately attached to the upper two-thirds of the ganglion. The posterior root was sectioned and the tumor was removed. The patient recovered and has been entirely free of pain. The tumor is a ganglioneuroma, containing a few nests of abnormal ganglion cells and showing large fields packed with the fibrils of spongioblastic cells.

Ganglioneuromas are not as common in the brain, as they are on the cranial or spinal nerves and by many, they are thought to represent heterotopias. They contain a variety of cellular elements, including adult nerve cells which, though definitely abnormal in position and in staining qualities, do show Nissl bodies and some tigroid material normally found in ganglion cells. The predominating glial elements are uni- and bipolar spongioblasts and with them may be varying amounts of neurilemmal cells and a fibrillary groundwork which may be difficult to identify. Parts of the tumor may be almost completely lacking in cells and appear to be a mass of fine interlacing fibrils. In our case of cerebral ganglioneuroma there were some foci of active neuronophagia, where the cells were undergoing a fragmentation of the nuclei and a fatty degeneration of the cytoplasm.

Neuroblastoma.—Like the ganglioneuromas, the *neuroblastomas* contain a large percentage of neuronal elements as well as certain glial components. We have had no example of this tumor in our series. It should be stated that though Cushing originally included three of these tumors in his series, later they were re-studied and reclassified either as medulloblastomas, neuro-epitheliomas, or as unclassifiable gliomas. Further study of a tumor which we believed to be a neuroblastoma proved it to be an astrocytoma.

The neuroblastoma is a rare tumor and possibly does not deserve classification by itself. It may be that it should be considered as a variant of the medulloblastoma. It is a cellular tumor, made up of non-polar or unipolar neuroblasts, which are characterized by their large clear nuclei and clear-cut nucleolus. These neuroblasts may resemble the large pineal cells of a pinealoma, since with a good gold stain the same sort of blunt process is frequently demonstrated on the young neuroblasts, but they differ in that they contain no blepharoplasten. All reported neuroblastomas have been found in the cerebrum.

Medullo-epithelioma and Neuro-epithelioma.—Neither one of these two rare gliomas has been found in our series of verified intracranial tumors. The *medullo-epithelioma* is a particularly primitive

and malignant type of tumor and it is made up essentially of undifferentiated medulloblasts. They are not quite the same cell, however, that one sees in a medulloblastoma, for they are larger and clearer than the ordinary undifferentiated medulloblast; the nuclei may be oval to spindle-shaped, and they have an epithelioid appearance. They tend to mass themselves about blood-vessels and many mitoses may be seen in any high power field. They arise from the original medullary epithelium and, therefore, are in the class of "midline," or "roof-plate" tumors.

The *neuro-epitheliomas*, also highly malignant, have been but rarely reported. They are made up of primitive spongioblasts which frequently arrange themselves ring-like about small open spaces and between such formations the richly cellular tissue bears a close resemblance to the medulloblastomas. Special stains will show a fairly generous amount of reticulin streaming from vessel to vessel through the cell masses. These tumors are to be found in the brain or spinal cord but predominantly in the cord, whereas the medullo-epithelioma has been reported found only in the brain. Both of them bear a close histogenetic relationship to the various retinal and sympathetic nervous system tumors.

THE MENINGIOMAS

Meningioma is the term proposed by Cushing for a group of intracranial tumors, of common origin, which may be found reported in the literature as dural endotheliomas, fibromas, sarcomas, psammomas, arachnoidal fibroblastomas, meningeal fibroblastomas, or combinations of these and other names. The name, *meningioma*, has come into fairly general acceptance, however, because it conveniently covers this large group of tumors of similar symptomatology and appearance, even though the exact histogenesis of these growths is still a matter for disagreement among neuropathologists.

The meningiomas are benign tumors which comprise between 13 and 15 per cent of all intracranial tumors. They do not invade the brain tissue in the sense that a glioma invades it, but they involve it only by their pressure upon it, and from that standpoint they are the most favorable of all intracranial tumors for surgical attack They may be extremely foreboding, however, and they offer the most difficult surgical technical problems for successful removal, mainly because of the enormous size to which they may grow before coming to operation. Because of their own great vascularity and that of the adjacent brain tissue, and because of their tendency to involve the major dural venous sinuses, their removal may entail a great deal of hemorrhage It is, therefore, highly desirable to prepare

the patient routinely for a blood transfusion before any attempt is made to remove a meningoma and, in our experience, their removal frequently requires a two-stage operation. The postoperative care of patients with meningiomas requires a high degree of sound surgical judgment to avoid the complications of a secondary edema, of convulsive seizures which sometimes occur due to irritation at the site of tumor removal, and of the shock which may develop as the result of the sudden removal of a large space-occupying mass from the intracranial cavity. Removal of the mass must be a painstakingly complete one, for if a small nest of viable tumor cells be left behind, these apparently benign lesions may recur with great rapidity. It is not the rule that signs of greater malignancy are present in such recurrent tumors, but the fact that a re-growth may occur and that some types of meningomas appear to bear a relationship histologically to the sarcomas, makes a complete removal imperative.

The gross appearance of meningomas is sometimes very startling. They may vary from a small, pea-sized nodule which would appear incapable of producing symptoms, to a large, meaty, nodular mass larger than a baseball. They have been reported as large as 400 grams and the largest one in our series weighed 165.7 grams. Davidoff[1] has reported the removal of a tumor weighing 800 grams, including the thickened bone which was removed along with the soft tumor mass. Cushing and Eisenhardt[2] report at length upon a patient upon whom 11 operations were performed over a period of twelve years and from whom a total of 1350 grams of meningiomatous tumor were removed, a mass equal to the average total weight of an adult brain.

In shape, the meningiomas may be divided into two groups: (1) massive, roughly spherical, lobulated tumors, and (2) flat, slightly elevated, irregular sheet-like growths. The majority of the globoid meningiomas are firm in consistency and have a thin capsule on the surface of which there are numerous large blood-vessels. Depending upon their location and direction of growth, their attachment to the dura mater may be large or small. They may be fairly soft, compressible and internally spongy with blood-vessels and large sinuses, but they ordinarily are firm enough to hold sutures which may be passed through them to aid in their removal. Their freshly-cut surface may vary from a yellowish-white cartilaginous tissue to a softly granular, sometimes cystic area of tumor. The second type, meningiomas en plaque, are ordinarily less firm; a

[1] Davidoff, L. M.: Meningoma—Report of an Unusual Case, Bull. Neurol Inst., New York, 6, 300, 1937.

[2] Cushing, H., and Eisenhardt, L.: Meningiomas, Springfield, Ill., Charles C Thomas, 1938.

capsule is frequently not demonstrable and they ordinarily are more vascular than the globular type. They are characteristically found arising from the basilar dura where their sheet-like extensions may preclude any attempt at a complete removal and where the underlying bone may be either greatly eroded or hypertrophied, but in any event it is invaded with tumor cells and is unusually vascular.

In their growth these tumors, particularly those of spherical shape, gradually bury themselves in the brain. The cortex, covered by pia mater and arachnoid, may be so dislocated as to give the impression that the tumor had its origin outside the arachnoid. As the tumor grows, the brain accommodates itself and the bed of the *growth may consist of convolutions which are greatly flattened,* atrophied, or actually destroyed by pressure. In the softer of the meningiomas the sulci of the brain may be invaded by small nodular outgrowths.

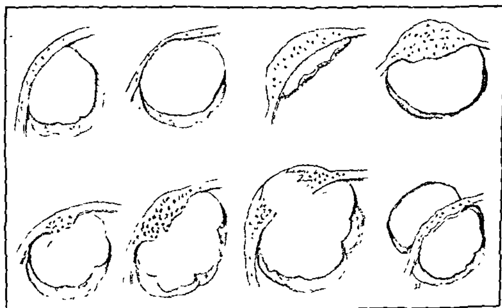


Fig. 65 — Diagrammatic drawing showing the changes which may be produced in the bone overlying a meningioma (After Cushing.)

The clinical course of the meningiomas is usually a long and slowly progressive one so that more often than not, the tumor has grown to an unusual size before it is recognized and the patient referred for surgical treatment Many times they would go unrecognized much longer than they do were it not for rather characteristic changes which they produce in the overlying bone which are recognized roentgenologically (Fig 65)

The bone of the skull in juxtaposition to a meningioma may show either erosion or thickening As a rule the spherical tumors produce

hyperostosis and generally, the bone overlying a flat meningioma may become extremely thin with the tumor near the surface yet not penetrating the dura mater. There seems to be no relation between the bony changes and the cellular structure of the meningioma. Very cellular tumors may be present with bone proliferation, yet fibrous tumors may be found with an extensive destruction of skull. When the skull is eroded over a meningioma, it presents a roughly circular, localized area of thinning which in the roentgen-ray film presents a mottled, spongy appearance in contradistinction to the clean-cut, punched-out defect of a metastatic carcinoma.

Derived as they are from the inner layers of the dura mater or from the arachnoid, columns of tumor cells may be found to pass through the dura along the course of blood-vessels. Tumor cells are crowded through the vascular dural spaces into the canaliculi of the bone. Absorption and the laying down of new bone begins. The tumor cells first appear in the diploë, then in the internal, and finally in the external table of the skull. Destruction of bone on the internal table leads to irregular jagged excrescences of bone surrounded by tumor. As the external table of the skull is absorbed and rebuilt, the outer surface of the skull gradually becomes elevated so that it slopes up onto the advancing prominence of the tumor. Tumor cells may be seen to fill completely the Haversian canals and spaces. A loose connective tissue which contains a few thin-walled blood-vessels separates the tumor filled bone trabeculæ from a surrounding thin, compact layer of fibroblasts.

In an interesting microscopic study of these bony changes, Penfield calls attention to the fact that the cells which form a thin strip of many layers over newly formed areas of bone have elongated nuclei which become oval and shorter as bone is approached. These nuclei are less closely packed than those of the tumor so that the tissue stains less deeply. Penfield states that these cells have to do with bone formation and that they resemble osteoblasts which are seen in ordinary bone repair. The osteogenic cells, which resemble fibroblasts derived from periosteum and bone tissue during ordinary bone regeneration, arise from either the outer layers of the dura or from the bone itself. As the growth of the tumor progresses, it produces rarefaction of the bone immediately overlying it, but it also stimulates bone-forming cells to proliferate and to lay down new bone.

Active over a long period of years the hyperostoses produced by these tumors may assume enormous and deforming proportions. At the apex of the tumor there is invariably a pad of tumor tissue where bone proliferation is most active. Newly formed bone spicules laid down perpendicular to the skull may be found surrounding a central area of absorption in the hyperostosis which is occupied by tumor cells. These views of the production of hyperostoses by the meningiomas are generally held although the usual earlier interpretation was that the growth of the underlying tumor stimulated the bone, without recognition of the fact that the bone becomes infiltrated by tumor cells Kolodny believed that the proliferation of bone in the skull is the result of an early and slowly progressive dilatation of

the blood-vessels in portions of the bone overlying the meningioma. The subsequent infiltration of the bone by tumor cells leads to bone destruction. He points to the fact that new bone is formed in layers parallel to the surface of the skull and that when bony spicules are present their course is determined by the course of blood-vessels and bands of connective tissue.

The difficulty of a satisfactory classification of the meningiomas has been of foremost concern since these tumors were first recognized as belonging to a distinct group. Mallory,[1] Penfield[2] and others have long believed them to be fibroblastic in nature, and so refer to them as "arachnoidal fibroblastomas" and "meningeal fibroblastomas." Among various French neuropathologists, especially Oberling and Roussy, the meningiomas are believed to be neuroectodermic in origin rather than mesodermic, so that they would necessarily, by this embryological beginning, be classed as a type of glioma. Cushing and Eisenhardt state: "Meningiomas, nevertheless, however qualified, are unmistakably direct descendants of the same mesothelial mother-cell, the meningocyte. Whether this cell, genetically speaking, is epiblastic or mesoblastic is disputed, but it begins to be reasonably certain that meningiomas and neurinomas are at least cousins with a common epiblastic paternity." Elsberg[3] has pointed out that though the similarity of the structure of the arachnoid cell clusters and the meningiomas points to their relationship, some meningeal tumors have no connection with the arachnoid. For example, meningeal growths often develop over the convexities of the hemispheres, at some distance from the dural venous sinuses. He suggests that these growths are derived from misplaced mesenchymal cells and that, therefore, they may originate from the dura mater, arachnoidea or pia mater In any event, even though there may be one common origin for the meningiomas, several different members of the family may be recognized by certain variations in cell type, architecture, staining qualities, reticulin and collagen formation, and content of mesenchymal, meningothelious, fibroblast'c, or angioblastic tissue. To add to the complexity of their histology, variations occur not only from tumor to tumor but within the same tumor, so that to arrive at a true understanding of the histology of any given tumor one should study several blocks from different sections of the tumor. Perhaps the most inclusive classifica-

[1] Mallory, F B The Type Cell of the So-called Dural Endothelioma, Jour Med Res, 41, 349, 1920

[2] Penfield, W The Encapsulated Tumors of the Nervous System, Surg, Gynec and Obst, 45 178, 1927.

[3] Elsberg, C A The Meningeal Fibroblastomas on the Under Surface of the Temporal Lobe and Their Surgical Treatment, Bull Neurol. Inst. New York, 2, 95, 1932.

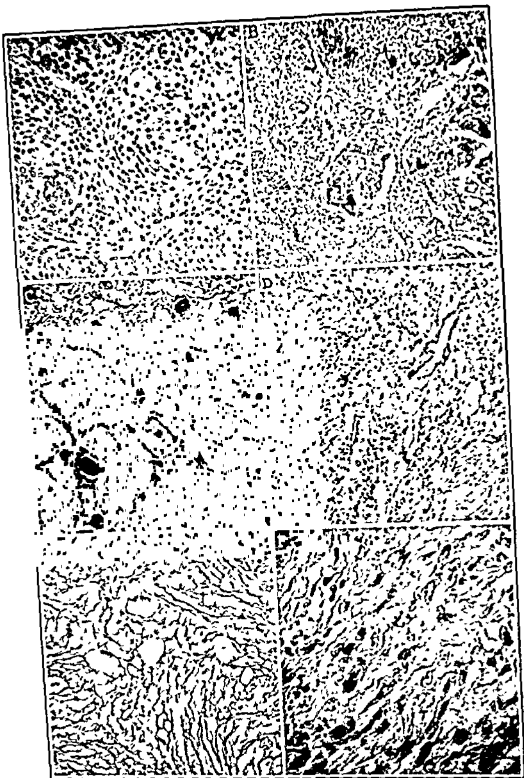


FIG. 66.—Photomicrographs of meningiomas illustrating the various types: (*A*) meningotheliomatous, showing a sheetlike appearance of cells of uniform structure. × 190, (*B*) fibroblastic, showing a large [illegible] the meningioma cells into nodules, × 97 [illegible] whorls of [illegible] (*D*) [illegible] formed blood-vessels, some of which are lined [illegible] higher magnification mitotic figures may be seen, (*E*) sarcomatous, showing the deposit of reticulin which is characteristic of this tumor, Perdrau stain, × 230; (*F*) sarcomatous, showing a variation in cell type, mitotic figures, and swollen fusiform nuclei, hematoxy[illegible] × 450.

tion, especially useful to the neuropathologist, is that which has recently been given by Cushing and Eisenhardt. But for convenience of ready classification with regard to both their gross and microscopic appearance, we have found the classification of Bailey and Bucy[1] very useful. The names of the various types of meningiomas in this classification are descriptive of the tumors. They are: mesenchymatous, angioblastic, meningotheliomatous, psammomatous, osteoblastic, chondroblastic, fibroblastic, melanoblastic and lipomatous To this might be added the sarcomatous type, not meaning, however, that the true meningioma is ever a form of true sarcoma. (Fig. 66)

So far as the histological appearance of the meningiomas is concerned, by far the commonest picture is that of a cellular tumor, the cells of which have round to oval nuclei which frequently stain heavily with hematoxylin and eosin. The cytoplasm is fairly abundant but very light staining. Such meningotheliomatous tumors may show only an occasional whorl arrangement of the cells, while others are almost completely made up of these nests of cells arranged like leaves in a whirlwind. Sometimes, such tumors have elongated nuclei which tend to arrange themselves in streams and palisades, so that upon first study an acoustic neurinoma may be suspected. Instead of the meningothelial cells, the predominating cell may be the fibroblast Many tumors show only an occasional darkly staining psammoma body, while others seem to be made up almost altogether of these fine gritty bodies to the exclusion of cells Bone and cartilage are frequently found in the larger solid, old, convexity meningiomas. Some tumors appear to be of a very loose structure, with angulated cells giving off irregular cytoplasmic streamers. Such tumors are not commonly vascular, nor have we found them to contain psammoma bodies and their general architecture, as well as cell type, justifies the name of mesenchymal meningioma We have had several angioblastic meningiomas in our series, and they have all shown the characteristic richness in fine vascular spaces, the walls of which are lined by a fine endothelium The neoplastic cells in this type, in contradistinction to these already named, frequently show mitotic figures and the Perdrau stain shows a rich deposit of reticulin Parts of the tumor may be mesenchymatous and the mistake may sometimes be made of confusing these tumors with hemangioblastomas The sarcomatous meningiomas likewise are reticulin producing. They show a variation of nuclear form, the predominating nucleus being fusiform or oval in shape, and mitotic figures are in rich abundance. These and the angioblastic meningiomas are definitely malignant and in our experience they

[1] Bailey, P and Bucy, P C The Origin and Nature of Meningeal Tumors, Am Jour Cancer, 15, 15, 1931

have shown a tendency for quick recurrence, even when apparently completely removed at operation. Melanin-bearing meningiomas are less common and we have found such deposits of pigment only in the meningotheliomatous type of tumor. Lipomatous degeneration of a meningioma, likewise, is not of common occurrence.

Irrespective of their exact histological origin, architecture and cell type, clinically the meningiomas have favored sites of development. Consequently, they produce characteristic clinical pictures which betray their presence and they may therefore be conveniently classed regionally.

Olfactory Groove Meningioma.—The meningiomas which arise from the olfactory groove on the ethmoid bone commonly produce a clinical syndrome characterized by bilateral anosmia, optic atrophy upon one side, and a papilledema on the other. As they increase in size, the frontal lobes are pushed upward and rather obscure mental symptoms may develop. In addition, slight weakness in the facial musculature may also be present upon the side opposite to the optic atrophy. One of our patients illustrates these symptoms very clearly. (Fig. 67.)

For seven years a housewife, aged fifty-one years, had been blind in her right eye and had been told that she had a primary optic atrophy. Her family had noted that she was facetious and depressed in alternate periods. Contrary to her usual self, her habits had become slovenly and she could not be trusted to carry on her household duties. She became very irritable and easily angered, so foreign to her disposition that her relatives finally urged her to be examined. There was a complete primary optic atrophy in the right eye and a left papilledema. A complete loss of smell and a left facial weakness were the only other objective symptoms.

Thickening of the floor of the right anterior fossa of the skull gave us the additional clue and a large meningioma was removed from the olfactory groove on the floor of the anterior fossa.

Cranial Nerve Foraminal Meningioma.—The cell clusters spoken of previously are said to be present about the foramina of exit of all of the cranial nerves Therefore, the tumors in this group may be subdivided extensively. However, the most commonly encountered tumors are found at the foramen of the eighth nerve, about the sheath of the optic nerve and about the Gasserian ganglion. They may, therefore, simulate acoustic nerve tumors from which they may be differentiated by the chronology of the development of symptoms. The tumors which arise from the arachnoid sheath of the optic nerve, even in the orbit itself, produce a unilateral, painless exophthalmos with a primary optic atrophy. The pain of trigeminal neuralgia may be simulated very closely by meningiomas which

involve the Gasserian ganglion, but as the growth progresses loss of sensation in the distribution of the fifth nerve serves to differentiate the condition from a typical trigeminal neuralgia.

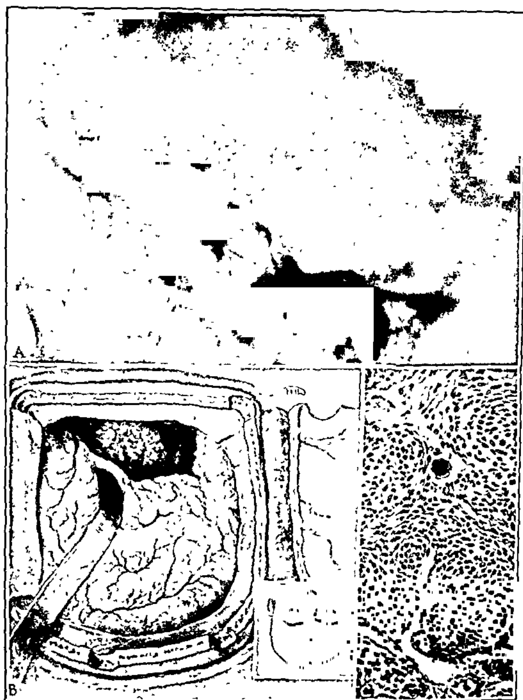


FIG. 67.—Olfactory groove meningioma. (*A*) roentgen-ray film which shows increased density of the floor of the anterior fossa of the skull; (*B*) drawing at operation which shows the location of incision and the position of the tumor beneath the frontal lobe, (*C*) photomicrograph of the meningioma which was removed, showing it to be of the meningotheliomatous type with psammoma bodies.

In one of our patients a meningioma which arose from the sheath of the optic nerve resembled in many respects an olfactory groove meningioma, except for the progressive exophthalmos. (Fig. 68.)

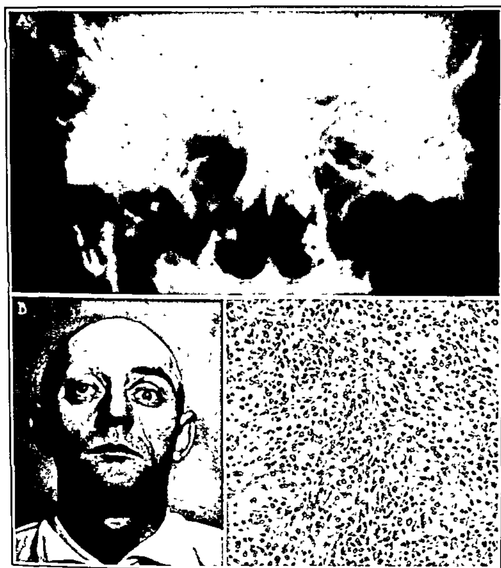


FIG. 68 —Cranial nerve foraminal meningioma: (*A*) roentgen-ray film which shows obliteration of the right frontal sinus by thickening of the bone due to the meningioma; (*B*) unilateral exophthalmus produced by the meningioma surrounding the right optic nerve; (*C*) photomicrograph of the meningioma which was of the meningotheliomatous type, × 200.

This patient rapidly lost the vision in his right eye and then noted that his eye began to bulge. Coincidentally, he had his first epileptic seizure which was a generalized convulsion without an aura. The right eyeball protruded about 3 mm. beyond the left. There was an optic atrophy on the right and a papilledema on the left. A left facial weakness was the only other symptom.

A large meningioma which surrounded the right optic nerve had grown forward and destroyed the roof of the orbit, pushing the orbital contents before it. The tumor was removed piecemeal with the electrosurgical unit.

Sphenoidal Ridge Meningioma.—There are several examples of this group of meningiomas in our series. They arise from the sphenoidal ridge which they commonly straddle, growing backward into the middle fossa or forward under the frontal lobe on to the orbital plate. Because of their location, they may produce epileptic seizures with an uncinate aura or they may produce a hemianopic field defect.

The long clinical course characteristic of the meningiomas in general is well illustrated by the following example of a sphenoidal ridge tumor. This patient also illustrates the similarity of symptoms produced by the meningiomas which occupy the anterior fossa of the skull irrespective of their origin and a still more important fact, that an incomplete removal of a meningioma is followed by a recurrence of the growth.

In 1919 the patient noted a failure of vision in the left eye and a slight protrusion of the left eyeball. Elsewhere, a flat meningioma was removed from beneath the left frontal lobe and it was thought that a complete tumor extirpation had been accomplished. Nine years later she developed Jacksonian epileptiform seizures and failing vision in the right eye. The exophthalmos of the left eye had progressed and there was a complete left optic atrophy with a right papilledema. Weakness was present in the muscles of the right side of the face and in the right arm. At operation a large tumor mass which straddled the left anterior sphenoidal ridge was removed, but the tumor had grown forward to destroy the roof of the left orbital cavity. The patient did well until 1940 when she rapidly showed signs of increased intracranial pressure; a complete right hemiplegia developed; she became comatose and succumbed undoubtedly to a recurrence of the growth.

Parasagittal Meningioma.—The meningiomas in this location arise from the wall of the superior longitudinal sinus and as a result may be attached to the falx cerebri as well. They may be characterized by a history of Jacksonian seizures which begin in the foot. Like all meningiomas which have a long history, in the course of years a spastic paralysis of one or both legs may develop. The skull over the tumor may show a characteristic hyperostosis or erosion which aids materially in their recognition. If, however, they arise anterior or posterior to the motor area, they may be exceedingly difficult to recognize

We have had one patient whose parasagittal meningioma produced a gradual loss of power in her lower extremities over a period of two years Upon admission to the hospital she was unable to walk and complained bitterly of a severe headache which was accompanied by vomiting As is true in many patients with meningiomas, there was a definite history of a blow on the top of her head about three years previously, but just what the exact relationship is between

trauma and the growth of these tumors, no one can definitely state at this time.

Both legs were spastic, completely paralyzed, and all of the reflex changes of an upper motor neuron lesion were present. Over the vertex of the skull there was a dome-like elevation about 8 cm. in diameter at its base. It was situated slightly more to the right than to the left of the median line of the skull and projected about 2 cm. above the level of the skull.

The roentgen-ray films of the skull showed it to be enormously thickened and perpendicular spicules of bone could be seen to extend beyond the limits of the external table. (Fig. 69.)

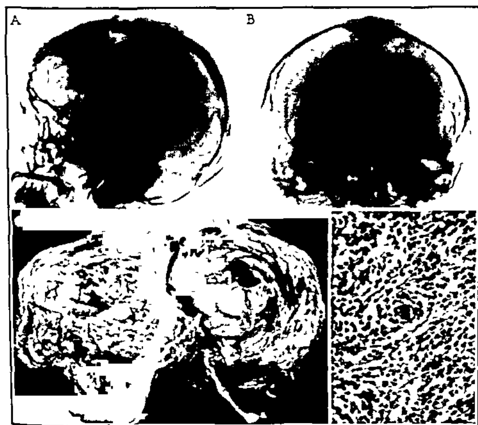


FIG. 69 —Parasagittal meningioma: (*A*) roentgen-ray film which shows a hyperostosis produced by the tumor on lateral projection; (*B*) on antero-posterior projection, (*C*) gross appearance of the specimen which shows its attachment to the falx cerebri and the sagittal sinus (removed at autopsy) (*D*) photomicrograph of the meningioma which was of the fibroblastic type

The day before operation the patient was found dead in bed and at autopsy a large meningioma was found attached to the superior longitudinal sinus. This structure passed through the center of the tumor mass which was bilobed. The cortex of each cerebral hemisphere was pushed aside and deformed by the growth which straddled the midline.

Sylvian Fissure Meningioma.—The flat type of meningioma is most frequently found overlying the Sylvian fissure. It probably arises from the sphenoidal ridge rather far lateralward. Since this type of tumor commonly produces a hyperostosis, often a sizable thickening of the temporal bone may be felt or seen. Clinically, they may be accompanied by weakness of the facial musculature upon the opposite side, and if they are on the left side of a right-handed individual, aphasic symptoms may gradually develop.

These tumors are usually not difficult to remove in their entirety and in our series we have several examples. In every instance the patient has remained well after operation and has been returned to his or her former economic and social position in life.

Suprasellar Meningioma.—These tumors arise from the meninges which cover the sinuses about the pituitary diaphragm. Their clinical symptoms simulate very closely the pituitary adenomas and craniopharyngeal duct tumors. The outstanding differential characteristic is a primary optic atrophy with a bitemporal hemianopsia without the corresponding changes in the sella turcica one expects to find with an adenoma of the hypophysis. These tumors may reach an enormous size and project into the region of the third ventricle.

One of our patients with a suprasellar meningioma had noted failing vision for two years and upon examination could count fingers in the nasal half of the right visual field only. The right fundus showed a complete primary optic atrophy and the temporal field of the left eye was markedly contracted and the nerve head was pale. As might be expected, these tumors present difficult surgical problems and in this case a two-stage operation was necessary, but after a stormy convalescence she has remained well, with a considerable return of vision, for twenty years. (Fig. 70.)

Meningioma of the Falx Cerebri.—The falx cerebri may be the site of origin of meningiomas which are usually spheroidal in shape. When they originate on the lateral surface of that structure they resemble the group of tumors known as convexity meningiomas. They also have a close clinical relation to the parasagittal group of tumors. The lower extremity is involved early in this group and Jacksonian epileptiform seizures may be present for many years before serious thought is given to the nature of the lesion. We have in our group of verified tumors one which originated from the falx between the occipital poles and rested upon the tentorium cerebelli.

Meningioma of the Sinus Transversus and Sinus Sigmoideus. There are numerous arachnoidal cell clusters about the transverse and sigmoid venous sinuses in the posterior cranial fossa Meningiomas not uncommonly originate in these positions and often the bulk of the tumor mass is situated in the cerebellopontile angle.

When this is true, a mistaken diagnosis of an acoustic neurinoma may be made, but the chronological development of symptoms, so characteristic of an acoustic neurinoma, helps to differentiate them. Often the tumor may be situated partly above and partly below the

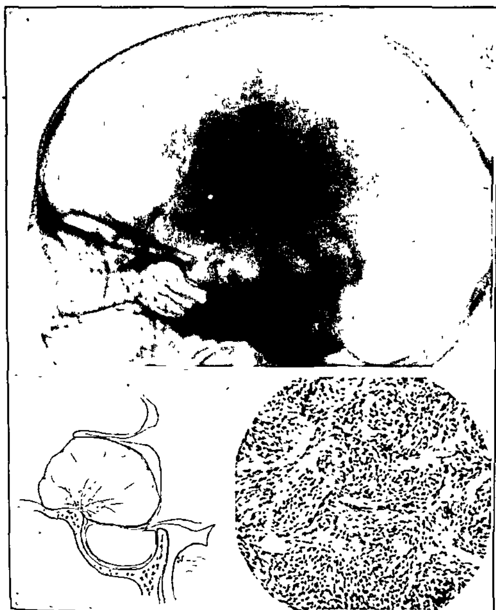


Fig 70.—Suprasellar meningioma (*A*) roentgen-ray film which shows the normal sella turcica in the presence of a suprasellar meningioma; (*B*) sketch which shows the location of the suprasellar meningioma; (*C*) photomicrograph of the fibroblastic meningioma which was removed.

tentorium cerebelli and then a combined occipital osteoplastic flap and a suboccipital craniotomy affords the best operative approach. We have recently operated upon a young woman who had a large and very vascular meningioma of the left cerebellopontile angle.

She had been seen elsewhere and because of her persistent vomiting and headache, she was given every available gastro-intestinal study. Finally, her gallbladder was removed. For some reason or another, her ataxia, deafness, slurring speech and papilledema were not accounted for and when she at last appealed for relief from her real lesion, her case was almost hopeless. In contrast, we operated upon



FIG. 71.—Convexity meningioma. (*A*) roentgen-ray film which shows the marked vascular changes in the skull overlying a meningioma of the convexity, (*B*) appearance of the patient upon discharge from the hospital, (*C*) photograph of the gross tumor removed at operation, (*D*) photomicrograph which shows the tumor to be of the meningotheliomatous type

a young man a few years ago who had few symptoms other than a persistent headache and a beginning edema of the optic discs. He was found to have a sarcomatous meningioma, of unusual vascularity even for a meningioma, well down along the brain stem and alongside the medulla. Surgical removal and the decompressive effect combined with a course of intensive deep roentgen-ray therapy.

have given him complete relief and he has returned to work completely free of symptoms.

Convexity Meningioma.—These meningiomas take origin from the arachnoidal cell clusters about the sphenoparietal sinus and the middle meningeal vessels. They are most frequently found over the frontal, paracentral, temporal, and parietal areas and are quite accessible. Often they produce focal motor convulsions which may lead eventually to a hemiparesis or paralysis. (Fig. 71.)

The fact that patients advanced in age are not exempt from intracranial tumors is illustrated by a woman, aged sixty-two years, who began to have frequent "cramps" in her right foot in 1925. These were followed by heaviness and stiffness of the right leg which interfered with her gait. Four years later she had a convulsive seizure which began in the right foot and spread to involve the leg and right arm, and many of these attacks were preceded by numbness in these extremities. Weakness developed in the right leg and arm so that for three months prior to hospital entrance in 1930 she was hemiplegic.

At operation a spherical firm mass could be palpated beneath the dura mater just posterior to the precentral gyrus This membrane was opened around the inferior edge of the tumor and the incision was then extended until the tumor was surrounded except at its medial edge. There it appeared to be attached to the dura mater near the falx cerebri. Silk stay sutures were placed in the mass and it was gradually tilted from its bed of flattened cortical convolutions. When it was freed, except at its attachment, the electrosurgical unit was used to coagulate the attachment and the vessels upon the surface of the tumor. The attachment was severed and a piece of dura beyond the point of origin and near the longitudinal sinus was removed with the tumor.

Basilar Meningioma en plaque.—We have had occasion to operate upon 2 patients with extensive sheet-like tumors which spread over a wide area on the base of the skull, and which were, therefore, not amenable to surgical removal. In neither case were we able to determine the origin of the tumor. In one case, the mass had invaded the bone of the middle fossa, sphenoidal wing, and roof of the orbit with a resultant unilateral exophthalmos of unusual degree and a complete blindness in the involved eye. The roof of the orbit was approximately an inch thick, so that an attempt at orbital decompression was unsuccessful This tumor was extremely vascular and was characterized by a rich deposit of psammoma bodies which occurred only in certain blocks of the tissue The other tumor, in a young man of twenty years, first gave clinical evidence of its presence by an involvement of the nerves to the extra-ocular muscles. Gradually, over a course of two years, every cranial nerve on both sides was involved in the spreading sheet of tumor, and he died a sudden death from hemorrhage in the nasopharynx which was due, most likely, to the eroding effects of the tumor which were noted upon roentgen-ray examination when he was first seen by us. Such tumors

She had been seen elsewhere and because of her persistent vomiting and headache, she was given every available gastro-intestinal study. Finally, her gallbladder was removed. For some reason or another, her ataxia, deafness, slurring speech and papilledema were not accounted for and when she at last appealed for relief from her real lesion, her case was almost hopeless. In contrast, we operated upon

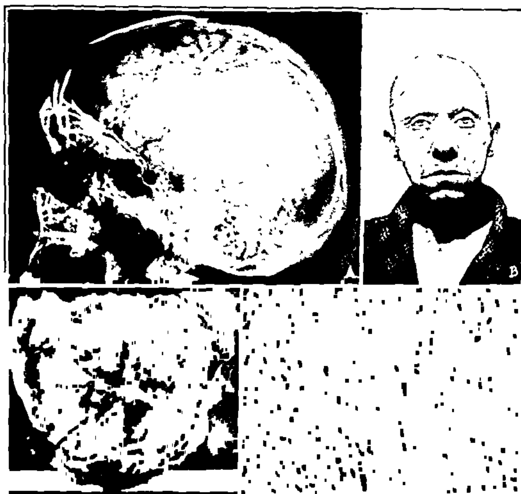


FIG. 71.—Convexity meningioma: (*A*) roentgen-ray film which shows the marked vascular changes in the skull overlying a meningioma of the convexity; (*B*) appearance of the patient upon discharge from the hospital; (*C*) photograph of the gross tumor removed at operation, (*D*) photomicrograph which shows the tumor to be of the meningotheliomatous type.

a young man a few years ago who had few symptoms other than a persistent headache and a beginning edema of the optic discs. He was found to have a sarcomatous meningioma, of unusual vascularity even for a meningioma, well down along the brain stem and alongside the medulla. *Surgical removal and the decompressive* effect combined with a course of intensive deep roentgen-ray therapy,

have given him complete relief and he has returned to work completely free of symptoms.

Convexity Meningioma.—These meningiomas take origin from the arachnoidal cell clusters about the sphenoparietal sinus and the middle meningeal vessels. They are most frequently found over the frontal, paracentral, temporal, and parietal areas and are quite accessible. Often they produce focal motor convulsions which may lead eventually to a hemiparesis or paralysis. (Fig. 71.)

The fact that patients advanced in age are not exempt from intracranial tumors is illustrated by a woman, aged sixty-two years, who began to have frequent "cramps" in her right foot in 1925. These were followed by heaviness and stiffness of the right leg which interfered with her gait. Four years later she had a convulsive seizure which began in the right foot and spread to involve the leg and right arm, and many of these attacks were preceded by numbness in these extremities. Weakness developed in the right leg and arm so that for three months prior to hospital entrance in 1930 she was hemiplegic.

At operation a spherical firm mass could be palpated beneath the dura mater just posterior to the precentral gyrus. This membrane was opened around the inferior edge of the tumor and the incision was then extended until the tumor was surrounded except at its medial edge. There it appeared to be attached to the dura mater near the falx cerebri. Silk stay sutures were placed in the mass and it was gradually tilted from its bed of flattened cortical convolutions. When it was freed, except at its attachment, the electrosurgical unit was used to coagulate the attachment and the vessels upon the surface of the tumor. The attachment was severed and a piece of dura beyond the point of origin and near the longitudinal sinus was removed with the tumor.

Basilar Meningioma en plaque.—We have had occasion to operate upon 2 patients with extensive sheet-like tumors which spread over a wide area on the base of the skull, and which were, therefore, not amenable to surgical removal. In neither case were we able to determine the origin of the tumor. In one case, the mass had invaded the bone of the middle fossa, sphenoidal wing, and roof of the orbit with a resultant unilateral exophthalmos of unusual degree and a complete blindness in the involved eye. The roof of the orbit was approximately an inch thick, so that an attempt at orbital decompression was unsuccessful This tumor was extremely vascular and was characterized by a rich deposit of psammoma bodies which occurred only in certain blocks of the tissue. The other tumor, in a young man of twenty years, first gave clinical evidence of its presence by an involvement of the nerves to the extra-ocular muscles. Gradually, over a course of two years, every cranial nerve on both sides was involved in the spreading sheet of tumor; and he died a sudden death from hemorrhage in the nasopharynx which was due most likely, to the eroding effects of the tumor which were seen after roentgen-ray examination when he was first seen by us. [illegible]

produce uncinate attacks. Symptoms of pressure upon the optic pathways may be late in appearing, however, and in our series we have a patient with a *very large, vascular chromophobe adenoma*, containing a great deal of dense connective tissue, who had a *widespread bony destruction about the sella turcica and body* of the sphenoid bone. Metabolic changes had been in progress for twelve years in this patient but he had complained of a narrowing of the field of vision and a decrease in visual acuity for only a little over a year. We have seen hypophyseal adenomas at the autopsy table so large that they have invaded the *temporal lobe, thus involving* the optic radiations and producing an homonymous hemianopsia. *Not infrequently these large adenomas press upon the floor of the* third ventricle and produce various hypothalamic symptoms.

Two *distinct clinical syndromes* are produced by the acidophile and chromophobe adenomas. The *acidophile adenoma* produces the condition known as acromegaly, or "hyperpituitarism," although a modification of this condition may be seen in patients whose hypophysis contains a non-tumorous proliferation of the acidophilic cells in a pituitary gland that is otherwise normal histologically. In individuals who have reached maturity before the disease begins, *the terminal ends of the bones enlarge, hence the name acromegaly.* If the disease process begins in adolescence before the epiphyseal *lines have closed, there is a generalized osseous overgrowth* and "gigantism" results. The changes which occur in acromegaly continue over a number of years to produce an appearance of the body which is so typical as to be pathognomonic The patient may become aware that he has to buy larger hats; that his hands and feet have become larger; that his fingers and toes are thicker and spadelike in configuration, and that his voice has deepened. The connective tissue undergoes hyperplasia which is noted in the enormous thickening of the lips, nose, scalp, tongue, glottis and subcutaneous tissue of the hands and feet. *The spaces between the teeth become wider* as the maxillary bones increase in size, the lower jaw enlarges until the occlusion of the teeth becomes impossible and the lower teeth protrude beyond the upper The chest becomes deeper and the patient seems to grow shorter as he becomes stoop-shouldered. The scalp may become so thick and wrinkled that it resembles the skin of a bulldog. The bones of the skull usually become greatly thickened but they are quite soft and vascular. In women, the earliest symptom may be a loss of the menstrual periods and the males lose their *sexual libido and quickly become impotent* The basal metabolic rate tends to be elevated and a glycosuria which is resistant to treatment *by insulin is not uncommon.* (Fig. 72.)

There is evidence to show that all of these symptoms in acromegaly

are the result of secretion from the acidophilic cells of the adenoma. But this is not true in the case of the *chromophobe adenoma*, in which the clinical picture is that of "hypopituitarism", for the cells have never been shown by any method to have secretory capabilities. Though the term, hypopituitarism, may not be entirely correct as a designation for the symptoms produced by the chromophobe adenomas of the hypophysis, it has sufficient accepted clinical usage to denote a definite syndrome. As in acromegaly, the first symptom of these tumors in the female is a loss of the menses. In the male, loss of libido and impotence are preceded by a complaint of fatigue and loss of energy. The hair over the face, in the axillæ, over the chest and on the pubis gradually thins and disappears; the skin becomes dry and very soft and has a waxy, pale color. Due to the loss of the beard and smoothness of the skin, such patients frequently



FIG. 72 —Appearance of a patient before and after the development of the symptoms of acromegaly; characteristic appearance of scalp (cutis vertices gyrata)

have a youthful facies not in keeping with their true age. There is an increase in the subcutaneous tissue, particularly over the hips, pectoral areas, and deltoid prominences in the male to such an extent that the body takes on a feminine appearance. The basal metabolic rate is almost invariably far below the normal range. (Fig. 73.)

In the mixed or "fugitive" type of hypophyseal adenoma, both chromophobe and acidophile tumor cells are present and, interestingly enough, the clinical evidence of both hypopituitarism and acromegaly may be present in these patients in varying degrees of prominence.

In addition to these symptoms of hypophyseal dysfunction and irrespective of their type, these tumors show certain other symptoms in common. Headaches develop as soon as the tumor grows large enough to stretch the dural capsule. The pain is dull and may be

described as a sense of pressure behind the eyes or in the temples. Quite often our patients have given a history of a severe headache of several years' duration which has suddenly ceased. In these patients, the tumor has usually ruptured through the dural capsule or has eroded its way through the floor of the sella turcica and thus, in one of these ways it has obtained more room for its continued growth. Besides the visual field defects produced by pressure of the tumor upon the chiasm, an optic atrophy results and if the tumor ruptures through the dural capsule and reaches such a size that its bulk



Fig. 73 —Photograph of a patient with hypopituitarism (Fröhlich's syndrome).

produces a rise in intracranial pressure, then a papilledema may be superimposed upon the atrophy.

The *basophilic adenomas* are not characterized by any local effect at their site of origin but are accompanied, rather, by a pleuriglandular syndrome consisting of hypertrichosis and amenorrhea in the female; painful obesity of the plethoric type; vascular hypertension; acrocyanosis and purple striæ of the skin over the trunk and thighs It has never been proved to be the result of the small, local

basophilic adenoma alone, for various other lesions may be found especially in the adrenal glands, pancreas, thymus and gonads.

Roentgenologically, hypophyseal adenomas produce a characteristic change in the sella turcica. The result of pressure from within the sella by an expanding tumor mass is to produce a "ballooned-out" appearance. The anterior clinoid processes become thinned and sharp and according to the direction of the growth, they may not be affected equally. In advanced cases they may become obliterated completely by pressure erosion. The floor of the sella may be thinned and depressed into the sphenoid sinus or may be

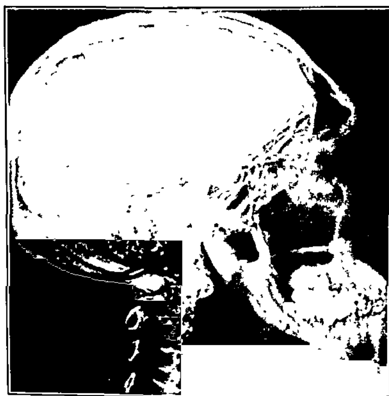


Fig. 74.—Roentgen-ray of the skull of a patient with acromegaly, which shows the enlarged sella turcica, thick bone of the skull and prognathism.

eroded through so that the fossa and sinus appear to be continuous. The important point to be emphasized is that the changes in the sella are those of a globular expansion from within. One can easily imagine a bursting lesion within the sella which has forced its bony walls to give away. This appearance is quite different from the evidences of erosion of the sella which are produced by pressure from without, the result of extrasellar tumors or a secondary internal hydrocephalus. (Figs. 74, 75 and 76.)

With specific stains for the various cellular components, it is quite easy to distinguish between the microscopic appearance of the hypophyseal adenomas, though an accurate diagnosis may be made by the simple hematoxylin and eosin stain alone. The acidophile adenoma shows no acinous architectural form and the cells are loosely scattered without particular arrangement. There is little or no vascularization and stroma formation. There is a considerable variation in the size and shape of the cells some of which are multinucleated and many of which are crescentic in form. Mitotic figures may be found. The cytoplasm stains faintly pink with eosin, and special stains reveal cytoplasmic granules which stain a brilliant red.

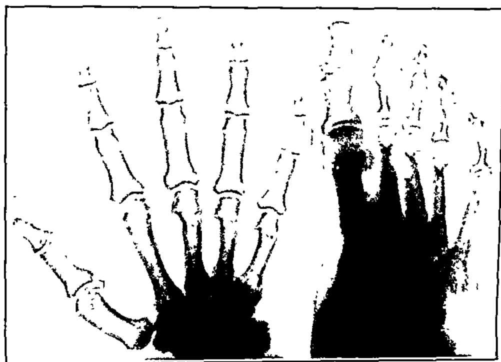


Fig 75 —Roentegn-ray films of a hand and foot of a patient with acromegaly which shows the characteristic enlargements of the ends of the bones.

These are the characteristic features of the cells. In the fugitive form of acromegaly the acidophilic tumor cells may show only a rim or halo of such granules and they may be mixed more or less evenly though in increased number among the other cells of the anterior lobe.

Not many basophile adenomas have been studied but they are characterized by an intra-hypophyseal nodule of microscopic size, composed of large, ovoid basophilic cells with dark oval nuclei and cytoplasm filled with coarse, deep blue granules. The structure of such a nodule is commonly acinous; the cells arrange themselves into many small ring-like alveoli.

The cells of the chromophobe adenoma are often grouped into masses or cords, delimited by small, stringy vascular sinuses. A true connective tissue stroma is rare, unless the tumor be one of quite long standing, wherein the connective tissue strands may be coarse, heavy and in places hyalinized. The cells have a fairly generous amount of light-staining cytoplasm in which no granules can be demonstrated. The nuclei are large and the amount of contained chromatin varies but is never large. Mitotic figures are very rare. There is evidence of pyknosis in some of the cells but most

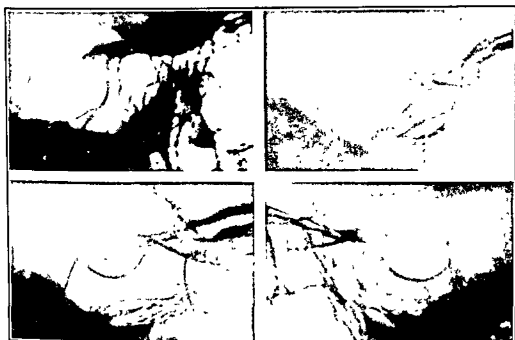


FIG 76 —Four roentgen-ray films which illustrate various types of destruction of the sella turcica due to an hypophyseal adenoma

cells have a plump nucleus and a distinguishable cellular outline. (Fig. 77.)

Surgically, one cannot cause the many symptoms produced by a glandular dysfunction to disappear by the removal of an hypophyseal adenoma. It is true that in many acromegalic patients a certain amount of subcutaneous edema disappears which improves the appearance decidedly but practically, the bodily changes are permanent. However, if a patient can be prevented from becoming blind, these glandular changes will not interfere with his or her return to a life of usefulness and independence. Consequently, at present the surgical treatment of hypophyseal adenomas is exactly where thyroid surgery stood in its early development. Adenomas of the hypophysis are removed to relieve pressure against the optic

chiasm just as thyroidectomies were performed to relieve pressure against the trachea. It is a pertinent question to ask why surgery should be postponed until irreparable damage has been produced, particularly if roentgen-ray evidence of a beginning enlargement of the sella turcica is present, though no visual field changes have made their appearance. At present the consensus is that visual field disturbances constitute the surgical indication in hypophyseal adenoma. There is an abundance of proof in every neurosurgical clinic that removal of these tumors not only prevents the gradual development of complete blindness but that in many patients the visual fields may widen out or they may even return to normal.

Realizing that at present there is no completely satisfactory form of substitutional therapy for patients with dyspituitarism and that many fractions of the secretion from the hypophysis have been described as having definite physiological activity, we have for several years been accumulating on all of our postoperative patients with or without replacement therapy, or on those patients with known tumors in whom there are no surgical indications, data concerning the thyroid, adrenal, renal, gonadal, pancreatic and hypophyseal functions, blood chemistry determinations and changes in neurological status. Recently, we have also studied the twenty-four hour urine output for 17-ketosteroids and the eosinophil response in these patients. It is hoped that such studies may lead to a better understanding of the proper time and method of both surgical and medical therapy for such patients. We have already found that though their headaches and visual field defects have been improved by operation, it is not uncommon for them to complain of increased perspiration, general weakness and mental slowness; an increase in the endocrine symptoms attributable primarily to the hypophysis; the onset of symptoms of a secondary active syndrome such as might be attributed to the basophilic cells of the pituitary; or, finally, other glands of internal secretion, such as the pancreas or thyroid, may show symptoms of dysfunction. We have observed 3 acromegalic patients who have developed a goiter and diabetes mellitus following the removal of an adenoma of the hypophysis In none of these patients before operation were there symptoms which pointed directly to the pancreas or thyroid glands.

In the early operations for hypophyseal tumors, they were removed either by a transsphenoidal or a transfrontal operation, but a careful comparison of the results obtained by the two operations has brought about the almost universal employment of a transfrontal approach Many patients had undergone previous intranasal operations which added greatly to the difficulties of a transsphenoidal operation; the operation was dangerous because the intracranial cavity was thus

exposed through a non-sterile avenue of approach; and, most important of all, many patients returned for operation because of a recurrence of their growth and thus eventually required a transfrontal operation. Moreover, many adenomas affect the chiasm by pressure but may never erode the floor of the sella turcica. Of course, those rumors which burst through the diaphragm sellæ and extend into

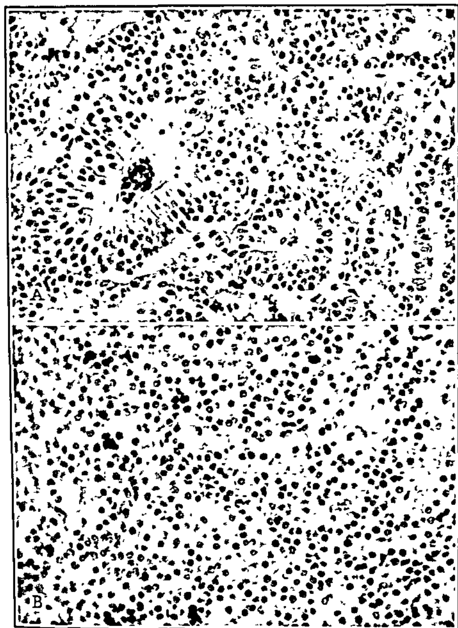


Fig 77.—Photomicrographs of hypophyseal adenomas: (*A*) chromophobe adenoma which shows the arrangement of the cells in cords around thin vascular sinuses and a scanty connective tissue stroma, × 365; (*B*) chromophile adenoma which shows the variation in the depth of staining of the nuclei which are of variable size and shape There is no characteristic pattern, vascular sinuses, or connective tissue stroma, × 380.

the cranial cavity cannot be reached completely by a transsphenoidal procedure.

In our group of 85 patients with hypophyseal adenomas, 35 have lived over five years; 10 have lived from two to five years; 2 have lived from eighteen months to two years; 3 have lived from twelve to eighteen months; 5 have lived from six to twelve months; 5 have lived from the time of hospital discharge to six months, and 12 patients died in the hospital following operation. One patient died without operation and was verified at autopsy. We have been unable to contact 12 patients, 1 of whom lived from two to five years and 1 lived from eighteen months to two years. Ten patients could not be traced immediately following their discharge from the hospital.

It is our custom to employ deep roentgen-ray therapy following operation and this is started as soon as the operative wound is well healed. We do not believe in the use of radiotherapeusis as a substitute for surgery. The danger of the development of serious impairment of the visual fields and even blindness following the edema produced by roentgen-ray therapy is a real one and certainly the tumor should be removed and the chiasm thus decompressed before such treatments are begun. Even with the advantages of an operation it is not uncommon for patients to complain of headache a few hours after a roentgen-ray treatment, for such treatment always causes a transient edema of the brain.

It is our custom to give a total of between 4500 and 5000 r units of roentgen-ray therapy to hypophyseal tumor patients following operation. We use heavily filtered radiation (200 k.v.—0 4 mm. tin, 0.25 mm. copper and 1 0 mm. aluminum filters) Five portals about 3 inches in diameter are used; frontal, occipital, vertex and 2 lateral and the number of r units is divided equally between them. Treatments are given five times weekly and at a distance of 20 cms the tube is always directed toward the hypophyseal fossa.

Cushing has summarized the matter succinctly when he said: "It may reasonably be assumed that in those clinics where radiation for these lesions is still routinely advocated, the surgical results have been poor and the mortality disturbingly high." In Cushing's large series of 349 patients with hypophyseal adenomas operated upon 403 times, there were 25 deaths, giving a 7.1 per cent case mortality and a 6.2 per cent operative mortality.

Endocrine Substitution Therapy.—A systematic study of endocrine function in patients who have had subtotal hypophysectomies has been made by Starr and Davis[1] with the intention of directing substitution therapy in the larger group of patients who have had

[1] Starr, P., and Davis, L.. Endocrine Studies of Patients After Subtotal Hypophysectomy, Ann Surg., 113, 778, 1941.

non-functioning tumors of the hypophysis, and of determining the necessity for further depressant therapy in those having hyperfunctioning pituitary adenomas. This was suggested by experience with patients after subtotal thyroidectomy, in whom a state of hypothyroidism, or rarely, persistent hyperthyroidism required continued supervision or treatment.

The symptoms of endocrine dysfunction, present before the operation, may continue postoperatively but should be susceptible to correction. If the pituitary deficiency is so pronounced that its tropic influence on the subsidiary glands is entirely lacking, the injection of pituitary extract is ineffective. On the contrary, when less serious pituitary deficiency exists, as in mild Froehlich's disease, the injection of pituitary extract may develop the target organ, make it more sensitive to pituitary hormones, and thus it is more effective. In many cases the postoperative pituitary remnant is not able to supply sufficient hormone to maintain the secondary gland. Furthermore, the deficiency has often been present for years and the atrophy of the receptive organ may be so great that it does not respond to extract injections. Hence it would seem that the object of endocrine therapy in these cases should be to supply the hormones of the glands which the pituitary normally maintains; that is, the thyroid, the adrenal and the gonads.

In the study of these patients, the basal metabolic rate and the blood cholesterol should be determined. The condition of the adrenal cortical function should be evaluated because the adrenal cortex is maintained by the tropic action of a pituitary factor. Carbohydrate balance, involving as it does the diabetogenic function of the pituitary, the responsiveness of the adrenal cortex to that hormone, the state of the liver as influenced by the thyroid and the activity of the insulin-producing organ, may be studied by the glucose tolerance and insulin tolerance tests. The gonadotropic function of the pituitary may be estimated by the history of amenorrhea and loss of sexual activity, combined with the physical examination of the sexual organs to determine the degree of atrophy. Frequently the vaginal epithelium was studied for evidence of estrogenic activity according to the morphology of the cells and their staining characteristics, after the method of Papanicolaou. Parathyroid function may be evaluated by a study of the blood calcium and phosphate and roentgen-ray films of the bones.

In the hypopituitary group, hypometabolism is the rule; 4 patients had extremely low basal metabolic rates, between −30 and −40 per cent; 11 were in the neighborhood of −20 per cent; this is the level usually found in hypophysectomized animals, and 2 were near the 0 per cent level. The blood cholesterol was significantly raised

in all 4 patients with the lowest rates, indicating thyroid deficiency; however, in only 6 of the larger group at −20 per cent was it elevated, while in the remaining 5 it was normal, indicating that in these cases the hypometabolism was not associated with relative hypothyroidism. Thyroid administration should be of value in 10 of these 17 patients to judge by hypercholesterinemia.

Adrenal insufficiency was suggested by abnormally high chloride concentration in the urine in 5 or the hypopituitary patients, and in one of the hyperpituitary group. Oddly, the 2 patients with the highest tests (as high as any found in fully developed Addison's disease) are in excellent clinical condition, carrying on normal lives. This suggests that in some way the hypopituitary state under the usual conditions of life of these patients avoids the sodium depletion of the body that leads to the crises which occur in patients with Addison's disease.

Consideration of the sugar tolerance curves brought out the expected lower curves; that is, greater tolerance to sugar occurred in the hypopituitary group, but under the conditions present the flatness was rarely pronounced and the curves usually were entirely normal. Hence this test in such cases would rarely be of diagnostic value in establishing the hypopituitarism which is certainly present The hyperpituitary cases, on the contrary, had a higher curve, that is, lower tolerance to sugar. This was most pronounced, of course, in the 2 patients with active acromegaly. Five of the 7 patients had sugar tolerance curves that were abnormally high

The insulin tolerance curves also brought out a distinct difference between the more and less deficient hypopituitary cases and the hyperpituitary cases. As was expected, the more hypopituitary patients had greater susceptibility to insulin. However, 2 of these patients maintained the blood sugar unchanged throughout the test In the hyperpituitary group, 3 of the 7 cases were unaffected by the insulin. One of these patients, however, had a marked insulin effect in combination with a high sugar tolerance curve.

Sexual function and the condition of the sex organs indicated great deficiency in all of the hypopituitary cases except 3. In these, normal gonadal development and function were maintained In the hyperpituitary group 2 were normal; 2 women, in addition, had amenorrhea and hot flashes indicative of pituitary activity characteristic of the menopause; 1 male of the 7 had impotence and atrophy similar to that of the deficiency group

Endocrine studies emphasize the loss of individual pituitary functions. These may be replaced by administration of the hormones of the glands normally maintained by pituitary activity, this is especially true of the thyroid Thyroid therapy, while indicated

and valuable, should be carried out cautiously. The objective should be the maintenance of a metabolic level correlated to the total function state of the hypopituitary organism. If the metabolic rate is −20 percent, then thyroid substitution should maintain normal metabolic processes at that level. Increasing the rate above this will place the hypopituitary patient in a state of relative hyperthyroidism detrimental to other physiological factors.

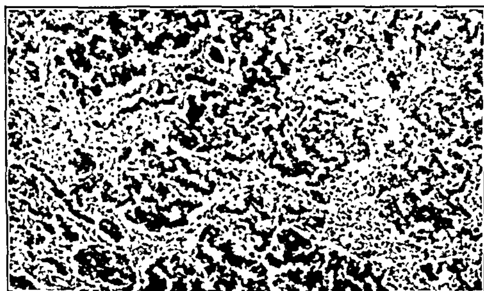


FIG 78.—Photomicrograph of adenocarcinoma of the hypophysis which shows the small, darkly staining nuclei of cells which are arranged in typical adenomatous alveoli. × 220.

Hyperpituitarism may continue after operation and be difficult to control or the acromegalic process may cease. The persisting disability may be due to hyperfunction of the subsidiary glands. The problem may become one of controlling hyperthyroidism, diabetes and hypertension. If this does not occur; that is, if treatment successfully stops the hyperpituitarism, the result is satisfactory and the energy production of the body and sexual function may become adequate.

Adenocarcinoma of the Hypophysis.—Adenocarcinoma of the hypophysis is one of the less common intracranial tumors and we have found only 4 cases in our series of verified tumors

They are primary tumors, originating within the gland itself, and they occur in individuals at or past middle age. It is common for these rapidly growing neoplasms to cause symptoms of hypopituitarism but ordinarily, these changes are less marked than in the more slowly growing chromophobe adenoma. Along with the

signs of pituitary dysfunction occurs the typical bitemporal hemianopsia of an expanding intrasellar lesion. Headaches are a common symptom and various paralyses of the extra-ocular muscles may appear late. Roentgen-ray studies show an enlarged sella turcica which maintains its proportions more accurately than it does in the presence of a chromophobe adenoma. It is customary, however, for both the anterior and posterior clinoid processes to appear eroded, greatly thinned, or decalcified.

Microscopically, these tumors are composed mainly of large, clear, atypical chromophobe cells with deeply staining nuclei. Many of the cells contain 2 or 3 nuclei and mitotic figures are in abundance. The cells are arranged in a typical adenomatous fashion, frequently forming tubules around the many small blood-vessels that are present. Acidophile cells are rarely found. (Fig. 78.)

We have treated these patients by surgical removal of the tumor, followed by intensive deep roentgen-ray therapy. Because of the highly malignant nature of these neoplasms, our longest survival period has been less than a year.

TUMORS OF THE BLOOD-VESSELS

These tumors comprise about 2 per cent of all intracranial tumors, though in our own series the frequency of occurrence has been higher. In conformity with others we have recognized two types of blood-vessel tumors. The *angiomas* are vascular malformations rather than tumors and present an hyperplasia of capillaries, veins, or arteries with an increase of the various cellular elements, but show no evidence of neoplastic disease. On the contrary, the *hemangioblastomas* are true neoplasms whose structure may be compared with embryonic hematogenous tissue.

We have observed 25 angiomas of the brain, 16 of which have occurred in the cerebral hemispheres and 9 in the cerebellum. The symptoms of the latter patients were quite indistinguishable from any tumor of the cerebellar hemisphere or cerebellopontile angle. The 16 angiomas of the cerebral hemispheres were all associated with epileptiform convulsions. Two were in the occipital lobe and visual hallucinations of light, so characteristic of occipital lobe lesions, were present. Vascular nevi of the face or scalp are very commonly associated with these vascular malformations of the brain. In one of our patients, a boy, aged eleven years, a large purple nevus occupied the cutaneous distribution of the ophthalmic and maxillary branches of the right fifth nerve. In addition, calcification of the angioma in his occipital lobe could be seen in roentgen-ray films of the skull (Fig. 79.)

Many of these patients complain of an intracranial bruit which may be heard if a stethoscope is applied to the skull. The sound can be abolished usually by compression of the carotid artery upon the same side.

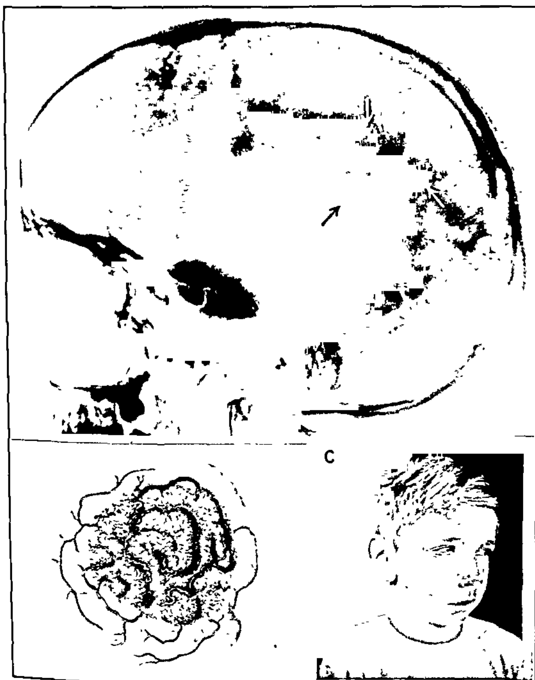


Fig 79.—Tumor of the blood-vessels (*A*) roentgen-ray film of skull which shows calcification in an occipital lobe angioma, (*B*) operative sketch of the angioma in the occipital cortex, (*C*) photograph of nevus in the distribution of the fifth cranial nerve on the same side as the angioma

Our cases fit into the classification proposed by Cushing in a monograph in which 16 angiomas were discussed. Rather simple *telangiectases* are characterized by an accumulation of numerous small capillaries lined by endothelium and separated by glia. *Venous angiomas* appear as simple or serpentine varices and a racemose type consisting of a tangle of large tortuous veins which may be situated superficially or may occupy the interior of a lobe of the cerebrum *Arterial angiomas* present arteriovenous communications, and since they originate from the pial vessels are usually superficially located. Many of these angiomas extend like a wedge into the cerebral tissue As in the venous angiomas the vessels are separated from one another by a firm glial scar.

At the operating table the venous and arterial angiomas resemble rather strikingly a huge tangle of earthworms. The telangiectatic type appears like a nevus of the face except that the individual small vessels are more distinctly evident. Surgically, the situation presented is a difficult one because in many, a wedge of the vascular malformation extends into the brain to a great depth. We have coagulated the small surface vessels of the telangiectatic angiomas, but deep roentgen-ray or radium bomb therapy may damage the vessel endothelium and produce obliteration with less danger to life.

We have operated upon 25 patients in this group of tumors with 1 postoperative death. In several cases we were sure of the preoperative diagnosis because of a very loud bruit or because of calcification visible in the roentgen-ray film. Six patients have lived over five years; five from two to five years; 3 have lived from eighteen months to two years, 1 from twelve to eighteen months and 2 from zero to six months. We have been unable to get in touch with 7 patients. Of this latter group 3 lived from two to five years; 1 from twelve to eighteen months and 3 from zero to six months. We continued roentgen-ray therapy in 1 young patient with a cerebellar angioma through a period of seven years, with increasing improvement in her symptoms and steady diminution in the intensity of her bruit until she succumbed from another cause.

Hemangioblastomas occur so characteristically in the cerebellum that such tumors described as occurring in the cerebral hemispheres may be looked upon with justifiable suspicion. The cerebellar symptoms produced are not peculiar to this tumor alone and as a matter of fact they resemble the cerebellar astrocytomas both clinically and upon gross examination at operation.

It was emphasized in the discussion of the astrocytomatous cysts of the cerebellum that a mural nodule of tumor should always be sought and removed. Whether the nodule will prove upon microscopic examination to be an astrocytoma or a hemangioblastoma cannot be foretold, although the former are by far the more common tumors.

Of our 38 examples of this type of tumor all have occurred in the cerebellar hemispheres and all have been cystic. Seven patients

died in the hospital following operation and 3 died in the hospital without operation. Nine patients have lived over five years, the longest survival period being twenty-one years; 7 patients have lived from two to five years; 2 have lived from eighteen months to two years; one from twelve to eighteen months; 3 from six to twelve months and we have been unable to trace 5 patients at this time but the last follow-up response showed that 1 lived over five years; one lived two to five years; one from six to twelve months and 2 had lived zero to six months following operation.

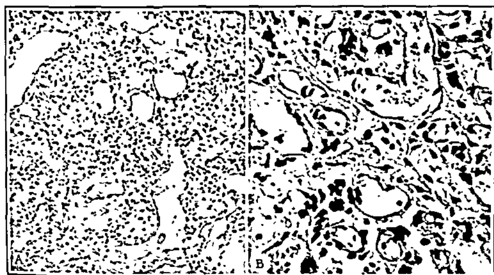


FIG 80 —Tumor of blood-vessels. (*A*) low-power photomicrograph of an hemangioblastoma of the cerebellum which shows many thin-walled vascular sinuses lined by true epithelium. × 160. (*B*) high-power photomicrograph showing flattened endothelium lining the vascular sinuses and a complete absence of mitotic figures. × 450

Hemangioblastomas which are unassociated with cysts do occur, but they may be so vascular that the slightest attempt to obtain a portion of the tumor for section causes serious trouble. When they are less vascular they may resemble very closely an astrocytoma and attempts at removal may be quite successful.

In 1922 Berblinger, and later Lindau (1926), called attention to the fact that these tumors may be associated with an angioma of the retina (Hippel's disease) and with cystic disease of the pancreas and kidney A retinal vein and artery may be large and tortuous, the artery beaded and both may be seen to enter an angioma at the periphery of the retina

Microscopically, the hemangioblastomas are made up of large epithelioid cells with an abundant cytoplasm and large nuclei filled with chromatin granules. There are many small and large vascular sinuses between solid masses of such cells, and these sinuses are lined

by elongated, darkly staining endothelial cells with flattened, vesicular nuclei. These sinuses are very thin-walled, and in places they are so numerous as to make the tissue appear honey-combed. The Perdrau stain shows a rich deposit of reticulin and affords an excellent means of quantitatively studying the sinuses. Areas of degeneration are common, and so-called pseudoxanthomatous cells may be seen in areas undergoing fatty degeneration. Inasmuch as this is not a malignant tumor, mitoses are not seen. (Fig. 80.)

The long survival period of patients with these tumors make them particularly amenable to surgical treatment. Like the astrocytomatous cysts, the hemangioblastomas were classed simply as cysts and their true history was unknown. In spite of this lack of accurate knowledge, removal of the cystic fluid alone gave relief which in many patients lasted for many years.

ACOUSTIC NEURINOMAS

Acoustic neurinomas constitute about 8 to 10 per cent of all intracranial tumors. They occur most frequently in adults of middle age, are slow growing, and originate from the sheath of the eighth cranial nerve. As they increase in size they produce symptoms of involvement of structures in the cerebellopontile angle

Clinically, the chronological development of symptoms produced by these tumors is so characteristic that the entity is one of the least difficult to identify Often the onset of tinnitus in the ear upon the side of the tumor may have been many years before the patient appears for examination. The noise may be that of hissing steam, a ringing bell, buzzing, or any one of many other descriptive terms may be applied to it. It may be continuously present or intermittent; but as it continues, hearing becomes affected and it is quite characteristic to have the patients state that they were first aware of a change when they found they were unable to hear well over the telephone. The tinnitus disappears as deafness becomes complete. As the tumor grows larger, other symptoms develop depending in the order of their appearance somewhat upon the direction in which the tumor enlarges. The patient is more likely to complain first of a peculiar feeling of numbness on the side of his face corresponding to his deaf ear, although his friend may have noted earlier a tendency for the corner of his mouth to droop, for saliva to escape from between his lips and his inability to close his upper eyelid completely Growth of the neoplasm medially compresses the cerebellar hemisphere and peduncles so that instability, a gait which resembles that of a drunken individual, true vertigo, and awkwardness of the hand and upper extremity on the same side gradually appear. Dysphagia and a

characteristic guttural and slurring type of speech are also common symptoms. Headaches, usually suboccipital in location, vomiting and papilledema occur the moment the tumor becomes so large that an obstruction to the flow of cerebrospinal fluid occurs.

The caloric and turning tests provide objective evidence of the partial or complete involvement of the vestibular portion of the eighth nerve in addition to the complete loss of hearing for the usual range of notes. Loss of the corneal reflex or a diminution of sensation over the distribution of the fifth nerve corroborate the subjective complaint of numbness. In a large number of patients, evidences of the relationship of these tumors to von Recklinghausen's disease are present, such as café au lait areas in the skin, pigmented moles, or even peripheral tumors in the skin. Unfortunately, the majority of patients present themselves for treatment when all of these symptoms are far advanced, mainly because their physicians have not recognized the significance of the combination of symptoms which have developed so insidiously.

For example, a patient, aged thirty-three years began to have a "buzzing and ringing of bells" in his left ear nine years before admission to the hospital. Within a year he was completely deaf in that ear, but it was not until seven years later that he began to have frequent attacks of muscular twitchings in the left side of the face which were followed by pain and numbness on the same side of his face. Six months before he came to the hospital he developed diplopia and so much awkwardness in his left upper extremity that he had to stop work. Headaches and vomiting were the final symptoms which made him seek further medical advice.

The roentgenological evidence of an acoustic neurinoma is based upon the demonstration of an enlargement of the porus acusticus. Towne has described a method of posterior projection which shows the petrous bones of both sides in profile and if this information can be obtained early in the development of these tumors, it will undoubtedly aid in their early diagnosis and surgical treatment. (Fig. 81.)

Grossly, these tumors are rather oval in shape and may vary from 2 to 8 cms. in length. As they enlarge they are bounded on one side by the petrous bone, above by the tentorium, and below by the floor of the posterior fossa of the skull. Medially and superiorly, they compress the cerebellar hemisphere and the brain stem. Often the latter may present a distinct concavity on its surface into which the tumor fits. The cranial nerves in the cerebellopontile angle may be engulfed by the tumor or are stretched tightly over its capsule. When these tumors are first exposed, a large subarachnoidal collection of cerebrospinal fluid lies over them and the inexperienced often mistake this for a cyst and the underlying tumor is neglected The

firm capsule of the neurinomas contains many dilated blood-vessels on its surface, but the yellowish, firm, rather nodular tumor tissue is usually not so vascular. The consistency of the tumor mass varies directly with the amount of fibrous tissue and fatty degeneration which is present. The superficial capsular vessels are not as harmless as they might appear to be because they usually originate from the



FIG 81.—Roentgen-ray film of the skull taken in Towne's position which shows destruction of the right porus acusticus due to an acoustic neurinoma

lateral aspect of the brain stem, to which the medial wall of the tumor is firmly attached. (Fig. 82.)

Microscopically, the acoustic neurinomas have been described as fibromas, sarcomas, neuromas, gliomas, endotheliomas, neurogliomas, fibrogliomas, and by various other combinations of terms. It is true that acoustic neurinomas vary from specimen to specimen and that blocks from different areas of the same tumor may show a very different microscopic picture. However, two definite types of tissue

are commonly identified. Some areas will show nuclei which are small, dark, and are arranged in a homogeneous mass of cytoplasm which has a loose, edematous appearance and which, in places, is obviously undergoing hyalinization. In such areas lipoid material will be found and clear, large, "foam" cells may be present in large nests. There is a moderate deposit of reticulin in these areas of degeneration. In other places, the cells are closely packed, even to

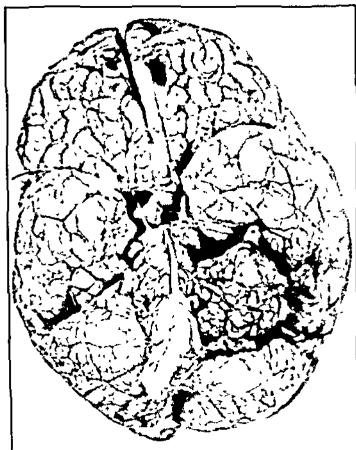


Fig 82.—Photograph of autopsy specimen which shows the location of a left acoustic neurinoma.

palisade formation; and the long, ovoid, or even spindle-shaped nuclei with their heavy stain stand out clearly in a background of streaming, whirling fibrils which look like fine hairs that have all been combed into the same arrangement. When such areas are cut at right angles to the long axis of the nuclei, the nuclei will appear small and round and the characteristic waves and bundles of fibrils will not be seen. There should never be cause to confuse the cell of an acoustic neurinoma with that of a fibroblastic meningioma or with a spongioblast, for the fibrils of an acoustic neurinoma are

extremely delicate, very fine in caliber, never show a hard, metallic, refractile quality and form waves which are made up of a great number of such fibrils. (Fig. 83.)

As Henschen first pointed out, the central segment of the eighth nerve is referred to as the glial portion and the peripheral segment as the non-glial portion. The glial portion has no endoneurium or neurilemma while the peripheral portion contains both of these structures and therefore resembles a typical peripheral nerve.

Skinner states that these tumors arise on the vestibular division of the nervus acusticus distal to the internal auditory meatus. He believes that the type of cell found is the neurilemma sheath cell and the fibrous tissue present is, in his opinion, in the nature of a tissue reaction. The particular histology of any given tumor therefore is dependent upon the inability or ability of the fibroblasts to confine the tumor cells.

The benign character of these tumors makes a total surgical extirpation a highly desirable procedure, but in our experience this is not as easily or as successfully performed as might at first be thought. Removal of the tough vascular capsule from the side of the pons and medulla is a highly precarious procedure, and, if successful, may leave the patient with a facial paralysis and a hemiplegia. Alternatively, one may split the capsule of the tumor with an electrosurgical scalpel and then remove the tumor piecemeal by scalloping and, if possible, by suction. The walls of the capsule may then be gathered together and coagulated without an attempt to remove it from its medial attachments. The age of the patient may well influence the surgeon in his attempt to remove a neurinoma completely. Extirpation of the tumor completely will produce a facial paralysis and may well cause symptoms of cerebellar dysfunction, or of other cranial nerve involvement, which cannot be rehabilitated. Endotracheal anesthesia and the upright posture during operation have proven to be of inestimable value in these operations. Removal of a portion of the lateral lobe of the cerebellum as a primary step in the operation is a helpful surgical procedure. Facial paralysis may be treated by a peripheral facial-hypoglossal or spinal accessory nerve anastomosis

We have operated upon 74 patients with acoustic neurinomas 83 times. One patient is alive twenty-four years, 1 has lived sixteen years, and 5 patients have lived ten years following operation. Eleven patients have lived over five years; 8 have lived two to five years; 4 have lived from eighteen months to two years, 3 from twelve to eighteen months; 2 from six to twelve months, and 6 patients have lived from zero to six months. Nineteen patients died in the hospital and 1 patient died before operation but was verified at autopsy Thirteen patients have not responded to our latest follow-up inquiries and of these 2 were alive over five years; 4 were alive two to five

years; 1 from twelve to eighteen months and 6 from zero to six months.

Cushing operated upon 171 patients 219 times with 25 postoperative fatalities, a 14.6 per cent case mortality and an operative mortality of 11.4 per cent. There is no doubt that every neurological



FIG. 83.—Acoustic neurinoma: (*A*) [illegible] showing fine reticulin fibers arranged [illegible] tudinally and transversely, × 220, (*B* [illegible] cigar-shaped nuclei arranged in parallel bundles, and an area of lipoid degeneration with fragmented nuclei and droplets of fatlike material, × 325.

surgeon passes through cycles of conservatism and radicalism in his attacks upon these tumors. If one has not been sufficiently radical in his first attack upon a neurinoma, a second operation need not be particularly dreaded.

CONGENITAL TUMORS

Craniopharyngioma.—Craniopharyngioma is a term used to designate that group of tumors which have been known as hypophyseal duct tumors, suprasellar cysts, Rathke's pouch tumors,



Fig. 84.—Patient with a craniopharyngioma. (a) seven days after operation, (b) one year after operation, showing the beginning symptoms of pituitary cachexia; (c) marked pituitary cachexia in the same patient shortly before death two and a half years after operation.

adamantinomas, and craniopharyngeal duct cysts. It includes all of those squamous epithelial intracranial growths which originate in the hypophyseal region and which may vary in structure from simple squamous epithelial lined cysts to tumors.

The hypophyseal vesicle or sac, which is a later stage of Rathke's pouch, is composed of stratified cylindrical epithelium. The great part of this tissue develops into the anterior lobe of the hypophysis. However, a single layer of this epithelium persists as a cleft which separates the anterior and posterior lobes of the hypophysis The hypophyseal duct, which is also a part of the pituitary anlage and which is lined by low cuboidal epithelium normally disappears It is in the location of the hypophyseal duct where this group of squamous epithelial tumors develop either from the anterior surface of the infundibulum or from beneath the capsule of the anterior lobe of the hypophysis Erdheim has demonstrated persistent fetal inclusions of squamous epithelium in these locations in a large percentage of normal hypophyses

The craniopharyngiomas occur most frequently in childhood or young adult life. They are almost invariably associated with the symptoms of dyspituitarism in the form of dystrophia adiposogenitalis or Froelich's syndrome. These children are fat and pudgy. In the males there is a distribution of fat characteristically female in type. The deltoid prominences are absent; the hips are wide; the thighs and legs are tapering; the pubic hair line is straight, and the prepubic pad of fat is prominent. In older individuals, and particularly is it noticeable in males, the distribution of hair over

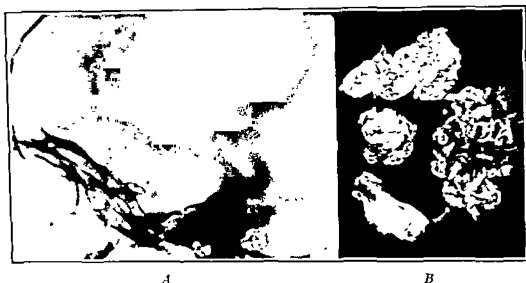


FIG. 85—Craniopharyngioma: (*A*) roentgen-ray film of skull which shows the suprasellar calcification in a craniopharyngioma; (*B*) photograph of tumor removed at operation, showing the marked deposit of cholesterin crystals in the cyst wall.

the body is scanty or absent. In adult life, the beard of these patients is fine and soft. Their skin is soft and velvety and appears to be pasty. The genitalia are infantile in type, which is to be explained by the development of the tumor in the preadolescent period of life. Almost without exception, the children who have such tumors have been very precocious mentally. (Fig. 84.)

In addition to the outspoken symptoms of hypophyseal dysfunction, these tumors produce symptoms of pressure involvement of the optic nerves and chiasm, stimulation or paralysis of important hypothalamic processes, and finally, hydrocephalus due to occlusion of the foramen of Monro. The optic nerve heads may show a primary atrophy or occasionally a papilledema may be superimposed upon a previously existing atrophy. The more solid types of craniopharyngioma, which are more common in the older individuals, produce visual field disturbances, such as a bitemporal hemianopsia, which

make it very difficult to differentiate them from an hypophyseal adenoma or a suprasellar meningioma. Diabetes insipidus, emaciation, somnolence, heat regulatory disturbances, and other hypothalamic symptoms are common. With the onset of hydrocephalus, headaches and vomiting are prominent symptoms and usually are the late symptoms which finally influence the patients to seek medical attention.

In each of the 22 cases in our series, calcification has been demonstrated in the roentgen-ray films. The deposits of calcium are delicate and spongy in appearance and may be seen in the midline of the skull either directly above the sella turcica or over the anterior clinoid processes. (Fig. 85.)

Craniopharyngiomas are usually found to be of one particular histological type, of which there are three general classes. The tumor may be only a small intrasellar simple cyst, the walls of which are lined with columnar epithelial cells, the contents being a mucilaginous, non-staining material. A commoner form is the solid tumor made up of heavy strands and arborizations of stratified, squamous epithelial cell masses. Between such masses is a coarse, connective tissue framework, which in places may have degenerated to a cystic or even hyaline state. Such areas of degeneration are frequently calcareous, or, they may contain a greasy, granular, dirty yellow material which is almost pure cholesterin. In large craniopharyngiomas these areas may be in a state of liquefaction and a dark, molasses-like fluid may ooze from the opened tumor capsule. Fat cells and loose squamous epithelial cells are present in abundance in such a fluid. The heavy masses of epithelial cells lining such cavities of degeneration may at times be of a cuboidal appearance and in certain foci arrange themselves into a stellate formation. Craniopharyngiomas with this architectural plan are loosely called *adamantinomas*, the third type of the tumor, because of the resemblance of these stellate cellular arrangements to the developing enamel organ of the embryonic tooth. (Fig. 86.)

In our own experience, there is no other group of tumors so difficult to operate upon as the craniopharyngiomas In the first place, the majority of these patients are poor surgical risks, often because of persistent vomiting and dehydration and many times because the children who are patients in this group are somnolent and moribund before they reach the neurological surgeon. One would expect that these cysts could be evacuated and their thin walls shelled out of their beds without much trouble. But the difficulties are that when evacuated and partially removed, they tend to reform and refill quickly or, and this has been our experience, symptoms of damage to the hypothalamus develop. Usually the latter occur very

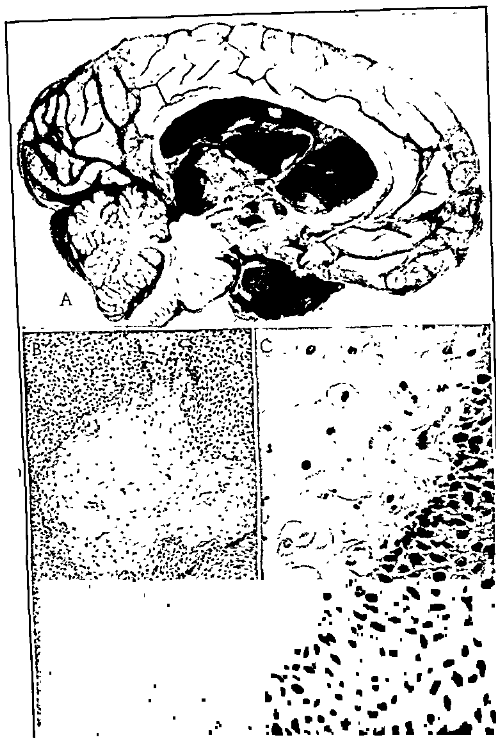


Fig 86 —Craniopharyngioma: (*A*) sagittal section of the brain showing the presence of a suprasellar cyst which has extended upward from the third ventricle into floor of the lateral ventricle; (*B*) photomicrograph of craniopharyngioma showing dense cords of epithelial cells surrounding small cyst, × 97; (*C*) photomicrograph showing detail of cyst wall which is formed by stratified, cuboidal epithelial cells; the cyst content is a homogeneous mass which contains free desquamated epithelial cells, × 475.

quickly following operation and one sees a hyperthermia develop when it has appeared that the operation was carefully done and that a disturbance of the hypothalamus was improbable. In one of our small patients, who was a round, fat, precocious youngester, practically blind when he was brought to the clinic, we succeeded in removing the cyst wall and a large amount of the yellow, glistening calcareous deposit. The boy made a fine recovery and was well for two years Then he began to lose weight and rapidly became emaciated, developed a diabetes insipidus, and soon succumbed.



A B

Fig. 87.—Craniopharyngioma: (A) photomicrograph of a craniopharyngioma of the adamantinoma type which was removed from an adult patient; (B) photograph of the patient four years after operation.

Our most successful case has been that of an adult male, who was rapidly losing his vision and who had many of the symptoms of hypopituitarism. The presence of very fine calcified areas just above the sella turcica led to the correct diagnosis. A cyst which contained thick, yellow syrupy fluid was removed and in the base of the cyst wall was a hard, firm, nodular tumor which proved to be an ademantinoma. This patient is active and free from symptoms thirteen years after his operation until his death from a coronary occlusion (Fig. 87)

The surgical story of these tumors, on the whole, is a discouraging and a baffling one. We have had 5 operative fatalities in 22 patients and each has been ushered in by a hyperthermia reaching 107° and 108° F. In Cushing's larger series 19 of the 87 patients succumbed after a total of 130 operations, giving a 21.8 per cent case mortality and a 14 6 per cent operative mortality

Chordoma.—Chordomas are very rare tumors and Cushing reported but 2 such neoplasms in 1932. We have seen 1 such growth

in our series of verified tumors and it occurred in a man of forty-four years, who withstood partial surgical removal of his huge tumor but succumbed seven days after operation. At autopsy an enormous, pink, jelly-like mass arising from the region of the body of the sphenoid bone was found to extend high into the midbrain and right cerebral hemisphere, destroying the major portion of the right temporal lobe. The fresh tissue was soft, moist, translucent and homogeneously of much the same appearance and consistency as dry granular gelatine which has been soaked for a few minutes in cold water. These tumors arise at the clivus, from the remains of

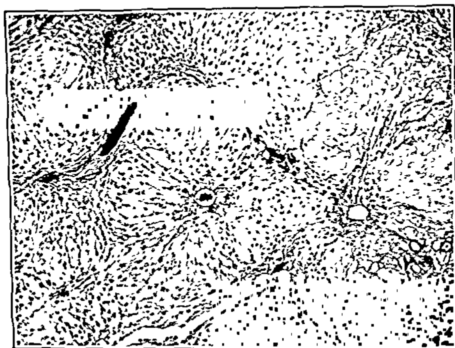


FIG. 88.—Chordoma. Photomicrograph which [illegible] of vacuolated cells wit[illegible] cells of notochordal [illegible] × 110.

the fetal notochord, and they may grow so large and erode the base of the cranial cavity so severely as to protrude into the nasopharynx.

It is generally believed that though these tumors are slow growing and slow to give signs of cerebral damage in keeping with their large size, they may actually be very malignant, as is evidenced by their propensity for invading bones and soft tissue, for re-growing rapidly after partial removal to even a greater size, and for metastasizing in rare instances.

Microscopic sections of our tumor showed large spherical to rhomboidal cells, arranged into clusters in a more or less mosaic pattern by means of a scanty connective tissue stroma. This stroma

passed into the substance of the tumor from the fairly well-developed connective tissue capsule and when the very rare blood-vessel was seen, it was very small and within this stroma. The large cells usually contained a mucinous, poorly staining material, or they even appeared vacuolated in many places. The eccentric nuclei stained poorly. No mitoses were found and blocks of tissue from various parts of the tumor all presented the same histological appearance. (Fig. 88.)

Cholesteatoma, Dermoid, Teratoma.—*Cholesteatomas* were described a century ago by Cruveilhier who named them "pearly tumors" because of the shiny, pearly surface of their outer membrane. They are of ectodermal origin and a true intracranial cholesteatoma is a rare tumor, for one must not consider in this group the commoner cholesteatoma of middle ear origin. True cholesteatomas occur along the midline in the middle or posterior cranial fossa. They are perfectly benign and of very slow growth. The outer layers of such a tumor are made up of flattened, "condensed", epithelial cells so stratified as to have lost most of their characteristics. The inner portion of the tumor is of a looser structure, with a peculiar honeycomb appearance of the transformed epithelial cells. The proper stains reveal the presence of much keratohyalin and cholesterin.

Dermoids are of rather rare occurrence intracranially and when found they are frequently of sub-pial location. Though they are more commonly intradural they are also found extradurally and even have been found between the two tables of the skull, under which circumstances the bone suffers a good deal of pressure erosion by the tumor. A favorite site for a dermoid tumor is within or below the vermis of the cerebellum. The cyst wall is made up of squamous epithelium in stratified to columnar arrangement. The contents may be hair, teeth, bone, sebaceous material, masses of squamous epithelium and a thick, tarry fluid rich in cholesterin. Fifteen cholesteatomas and dermoids occurred in Cushing's series of 2,000 verified tumors, whereas we have operated upon 6 dermoids and 3 cholesteatomas in our own series of tumors. Because of the benign nature of these tumors, a complete surgical removal brings about a satisfying permanent cure.

Four *teratomas* were reported by Cushing but there have been none of these tumors in our series. These rare neoplasms most frequently occur in the pineal, hypophyseal, or other anterior brain stem locations and their presence is manifested early in life. They have much in common with the dermoids so far as contents are concerned, for they contain bone, solid epithelial masses, neural elements, sebaceous material and cholesterin They are well encap-

sulated and usually well delimited from the surrounding brain tissue and could, therefore, be removed successfully were it not true that their size is usually quite large. Their location is a precarious one in that they are frequently attached by blood-vessels or meninges to neighboring brain tissue. It is not often that an attempt at surgical removal is followed by a successful outcome.

METASTATIC TUMORS

We have encountered 111 metastatic tumors of the brain, the majority of which have been verified at autopsy.

When metastases to the brain are multiple, surgical intervention is not indicated but often there is a solitary superficially placed metastatic lesion particularly in patients in whom the primary tumor is in the lung. The radioisotope dye test is valuable in determining this fact. The lung tumor often progresses slowly, and if the cerebral tumor can be removed, the patient may be benefited tremendously. We have had this occur in a patient who is socially and economically independent 14 months after his intracranial operation.

As might be imagined, there is no characteristic group of intracranial symptoms which stamp these tumors as entities. Symptoms referable to other systems, or a history of a previous operation for the removal of a tumor, make their recognition simpler. We have the impression though that patients with metastatic tumors exhibit a state of mental stupor and dullness which is entirely out of proportion to the objective neurological symptoms or the signs of increased intracranial pressure.

The radioactive isotope dye test has proven to be valuable in the detection of multiple intracranial tumor sites. The greatest concentration of the dye has occurred in the metastatic tumors.

Metastatic carcinomas are wild, erratic growths full of large, clear, reticulated carcinomatous cells, reproducing fairly well the architecture of the tissue in which the primary tumor arose. They furnish excellent opportunity for the study of the various stages of neoplastic cellular division, for mitosis in all the phases is to be seen everywhere. In the *metastatic hypernephroma* the identifying cell is large and has a clear cytoplasm, suggesting a fat cell. The slightly reticulated and abundant clear cytoplasm has a frothy appearance and close inspection reveals the presence of fine, dark evenly scattered granules. Cell boundaries are quite distinct. The nuclei are large, round or oval in shape, stain evenly and usually show no chromatin network. There is almost uniformly a dark, round centrally placed nucleolus. These cells fill up the spaces of a coarse reticular stroma in the tumor and between such masses of cells one finds other small,

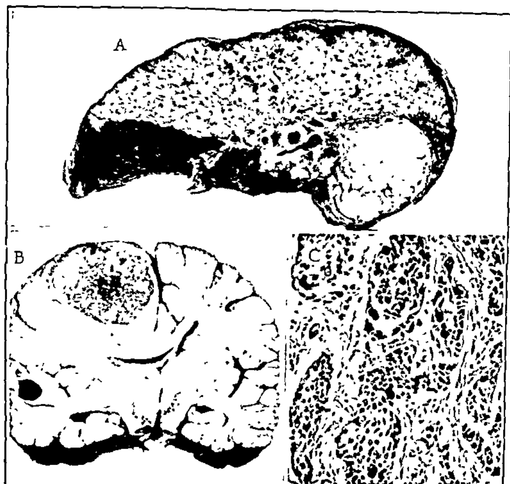


FIG 89.—Metastatic tumor (*A*) photograph of lung which shows the primary tumor, a bronchiogenic carcinoma,(*B*) the same tumor metastasized to the cortex of the brain, (*C*) photomicrograph of the cerebral metastasis which shows clusters of cells with darkly staining nuclei and many mitotic figures The arrangement of cells fairly accurately reproduces the microscopic appearance of the primary tumor of the lung, × 192

dense, darkly-staining cells with a poorly defined cytoplasm and no particular pattern or formation. *Metastatic sarcomas* may vary quite widely from tumor to tumor. Some may be melanin-bearing, while others are not. The cells vary from round to oval, the cytoplasm is characteristically not abundant and the nuclei stain fairly darkly. Such tumors are quite cellular, mitoses are frequent and large multinucleated cells are often found. The Perdrau stain shows a rich deposit of reticulin around the blood-vessels In many foci the cells arrange themselves in a ring-like, peritheliomatous formation and such an architecture is recognized by some pathologists as being the characteristic feature of a metastatic sarcoma. Of great use in the study of sarcomas, or other tumors of sarcomatous nature, is the Perdrau stain, which shows no cells but only the bundles of reticulin

which are characteristically thrown down by tumors of this type.

The primary tumor in our group of patients was located in the lungs, breast and intestinal tract, and 7 were hypernephromas. With

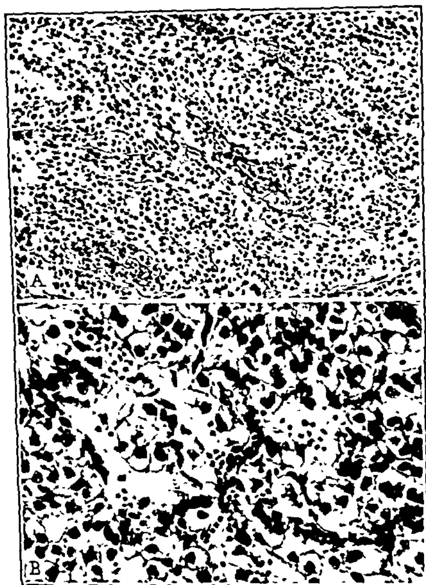


Fig. 90.—Metastatic tumor: (*A*) photomicrograph of metastatic sarcoma from the meninges of the brain. The primary tumor was a sarcoma of the spinal cord, × 226; (*B*) cerebral metastases from a hypernephroma showing cells with clear, slightly reticulated cytoplasm and darkly staining nuclei. All boundaries are distinct, and the cells are separated into clusters by thin-walled vascular sinuses, × 410.

the exception of these latter cases and 11 with metastatic sarcomas, the remaining 93 tumors, with one exception which we are unable to classify, have been metastatic carcinomas. Quite frequently the metastasis from a bronchiogenic tumor has been solitary and sur-

institute heliotherapy amidst favorable surroundings. Two of his patients thus treated were alive five and six years after operation and successive roentgenograms showed that the lesion had become progressively calcified.

For many years every intracranial tumor was regarded as a gumma until the patient was given long and extensive antiluetic treatment. We have never encountered a gumma and though 5 such tumors were found in Cushing's 194 verified cases up to 1911, only 11 were identified subsequently though ten times as many tumors were verified histologically.

These two types of granulomas each have a very characteristic appearance. The *tuberculoma* is a discrete lesion, set down with sharply defined limits in the surrounding brain tissue. It always shows an abundance of typical multinucleated giant cells, a degenerative ground substance, many gitter cells and masses of infiltrated lymphocytic cells. The *gumma* seems to be made up mainly of a necrotic, collagenous material which is filled with all sorts of tissue debris, gitter cells, plasma cells, macrophages and leucocytes. Many of the blood-vessels show thickened walls; many of the walls are ruptured and necrotic in appearance and small hemorrhages may appear alongside such a vessel. The characteristic feature is the perivascular lymphocytic infiltration which accompanies practically every vessel however small. (Fig. 91.)

MISCELLANEOUS TUMORS

Cysts.—Four interesting cysts of the brain have been encountered, two colloid cysts of the third ventricle, one cysticercus cellulosis due to Tænia solium, and one papillomatous epithelial cyst.

Colloid cysts are attached to the roof of the third ventricle and contain a gelatinous material which is quite homogenous. This is surrounded by a layer of epithelial cells outside of which is a layer of connective tissue. Clinically, they are difficult to diagnose, but an intermittent severe headache produced and relieved by sudden movements of the head may suggest the ball-valve action of such a tumor as it obstructs the cerebrospinal fluid pathway.

The *cysticercus cellulosis* specimen was identified at an operation for a cerebellar tumor. A milky, white gelatinous substance resembling the white of a slightly boiled egg was removed from the cisterna magna This patient was kept in the hospital and died seven months after his operation. Multiple cysts were found on the surface of the cerebral hemispheres and in the third ventricle.

At operation for what seemed to be a glioma of the parietal lobe, greenish brown turbid fluid was removed from the ventricle. It did

not coagulate on standing and contained red blood cells and other cell bodies with large nuclei which could not be identified readily. The cortex was incised and a large cavity containing similar fluid was opened into. Its walls were rugous and cauliflower-like. Microscopic examination revealed an unusual example of a *papillomatous epithelial cyst.* (Fig. 92.)

Osteoma.—We have observed 4 intracranial osteomas which have produced symptoms. One was a large bony mass removed from the inferior gyri of the parietal lobe with subsequent freedom from the convulsions which had brought him to our attention. Just what the etiology of this mass was, we are unable to say; but a hematoma or tuberculoma which became calcified and later ossified are perhaps the most likely possibilities.

We have never recognized an orbito-ethmoidal osteoma with intracranial complications though Cushing has described his experiences with these tumors very vividly.

Osteochondroma.—Only one such tumor has occurred in our series. It was a 280-gram mass removed from the left parieto-temporal region of a thirty-two year old woman. She recovered and was well fifteen years after her operation. The mass was white, nodular and hard and on microscopic section showed fields varying from pure bone to young cartilage and dense fibrous tissue. Staining properties were very poor and cellular boundaries hard to make out.

Meningeal Sarcomatosis.—This rare condition has been the subject of no small amount of misunderstanding and disagreement among neuropathologists. It is probably more common about the spinal cord and occasionally, a discrete sarcoma of the spinal cord may be accompanied by a diffuse sarcomatous growth in the meninges of the posterior cranial fossa. When such an invasion of the cerebellar meninges does occur, the condition is seldom accurately diagnosed because of the peculiar, varying, and unreliable clinical signs which are present. Frequently, the typical sarcomatous cells are melanotic and the tumor appears grossly like a spotty sheet of dark gray tissue which invades every chink and crevice where the leptomeninges attach to the cerebellum and medulla. Such sarcomatous invasion may occur in the dura mater as well and may extend anteriorward on to the undersurface of the pons and cerebral hemispheres.

The diffuse spread of the tumor and its close attachment to the brain precludes its complete surgical removal. It is highly malignant and broken off cells may seed themselves wherever the cerebrospinal fluid may float them. Recourse may be had to deep roentgen-ray therapy or to the radium bomb but the prognosis obviously is not very encouraging. We have had two cases of meningeal sarcomatosis of the leptomeninges one of which was a boy of four-and-a-half years.



A



B

FIG 92.—Papillomatous epithelial cyst (*A*) coronal section of the brain which shows a papillomatous epithelial cyst; (*B*) photomicrograph which shows islands of epithelial cells separated by a rather heavy connective tissue framework There are few blood-vessels in the tumor, and mitotic figures are rare.

Death occurred twenty-three days postoperatively and postmortem study revealed a spread of the plaques and nodules of tumor tissue over most of the surface of the medulla, cerebellum, pons and under-surface of the cerebral hemispheres.

UNCLASSIFIED TUMORS

This group represents those neoplasms which we are not, as yet, able to classify to our satisfaction. They are re-studied from time to time and they have been submitted to others for diagnosis. Often, a lapse of years may solve the problem when we obtain additional material from an autopsy specimen.

To us this group is a highly important one full of interest. We are firmly convinced that Cushing's efforts to correlate the clinical history and histological appearance of intracranial tumors has been a greater advance in tumor surgery than has been accomplished elsewhere in the body. If every general surgeon had verified the carcinomas of the stomach or breast operated upon by him, and had carefully correlated the life history of his patients, a great deal more would be known and less material which is now valueless would have accumulated.

Many of the intracranial tumors, particularly the gliomas, appear distinctly different in sections made from various parts of the tumor. This fact, many times, causes confusion among pathologists, but the clinical story aids in settling the question. The histopathology of intracranial tumors is still an open problem to which there must continue to be a common, pooled contribution of data and material.

THE ROENTGENOLOGICAL EVIDENCES OF INTRACRANIAL TUMORS

The evidence of an intracranial tumor which may be obtained by a direct roentgen-ray examination of the skull may be of considerable diagnostic aid, as has already been indicated.

Enlargement of the skull, most frequently the result of an obstructive hydrocephalus in children whose skulls are capable of expansion; convolutional digitations upon the inner surface of the skull, which are casts of the cerebral convolutions; and separation of suture lines are all signs of increased intracranial pressure. An intracranial tumor may be present, but these signs are not indisputable evidence; they are suggestive, but represent the effect of intracranial pressure from whatever cause.

The vascular channels in the skull may lead one to the correct diagnosis or they may be very confusing. The middle meningeal

artery channel may vary considerably in normal individuals and although in intracranial tumors it is common to find a marked dilatation of the diploic venous channels, most unusual vascular markings may appear symmetrically in a normal individual. Often a confluence of venous channels produces well-defined areas of decreased bony density. The same care must be exercised in evaluating these "venous lakes" as diagnostic criteria of a tumor.

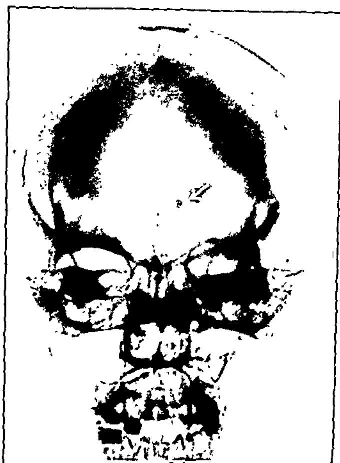


FIG. 93.—Roentgen-ray of skull showing shift of pineal gland to the left of the midline produced by a space-occupying lesion on the right.

The local areas of hypertrophy and erosion produced by meningiomas have already been mentioned. Often, pressure erosion of the margins of the foramen magnum may be demonstrated in cerebellar tumors.

Probably no two sella turcicas appear alike upon roentgenograms. The "ballooned-out" sella of an hypophyseal adenoma is characteristic, but often the pressure of a tumor from above, or a secondary hydrocephalus, may produce destructive changes in the sella which are difficult to differentiate.

A space-occupying lesion in the cerebral hemispheres pushes the falx cerebri and other midline structures to the side opposite the tumor. In about 50 per cent of all skulls the pineal gland shows calcification which is demonstrable roentgenologically. The pineal gland may be shifted, therefore, an appreciable distance to the opposite side regardless of whether the tumor is frontal, temporal, parietal or occipital in location. (Fig. 93.)

A deposit of calcium within the brain may occur as a physiological phenomenon in normal persons, or it may be found in neoplasms, hematomas, tuberculomas, aneurysms, chronic abscesses, areas of old meningeal disease and encephalitis. Calcification in the choroid plexus, pineal gland, or in the falx cerebri may be present in a normal brain.

Weed has classed the intracranial calcifications as punctate, vascular, trabecular, amorphous and periarterial. Psammoma bodies which have a definite external circular form, but no internal structure and corpora amylacea, in which concentric rings which simulate starch granules are present, belong to the group of punctate calcifications. Vascular calcifications often show a solid, cord-like appearance and an entire channel may be replaced by a calcium deposit. Trabecular calcifications are irregular, plate-like collections which are found in the trabeculæ of various tumors which have a fibrous framework. Amorphous deposits have no constant structure, but resemble débris. Periarterial deposits of calcium encircle arteries like a ring.

In addition to these rather general diagnostic aids, erosion of the optic nerve canals and the auditory meatus may be demonstrated by technical methods which have become quite simplified and are universally used.

Ventriculography and Encephalography.—As the result of his work upon the problem of hydrocephalus, Dandy conceived the idea that the most practical manner in which to determine the size of the cerebral ventricles and the point of obstruction to the circulation of the cerebrospinal fluid would be to replace the fluid by a medium which could be differentiated from the shadow of the surrounding bones of the skull upon a roentgenogram. In 1918, he suggested the use of air as a medium which would be non-irritating, non-toxic and readily absorbed and excreted. Dandy introduced air into the lateral ventricles after he had made burr holes through the skull and to this procedure he gave the name *ventriculography*.

He produced convincing evidence that the ventricular fluid could be replaced safely by air in the diagnosis of the level of obstruction in cases of hydrocephalus. However, in some instances in which no obstruction existed in the ventricular pathways and in which he wished to demonstrate the subarachnoid spaces, he could not completely fill the cisterns at the base of the brain by a ventricular

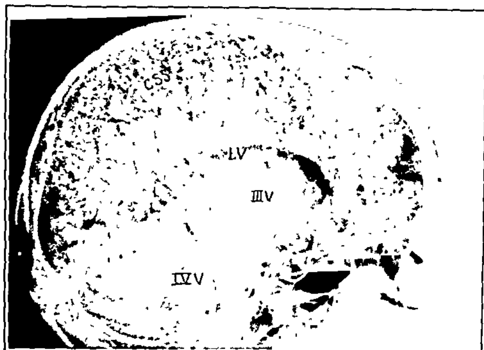


FIG. 94 —An encephalogram which shows the lateral ventricle (*LV*), the third ventricle (*III V*), the fourth ventricle (*IV V*), and the subarachnoid spaces (*CSS*)

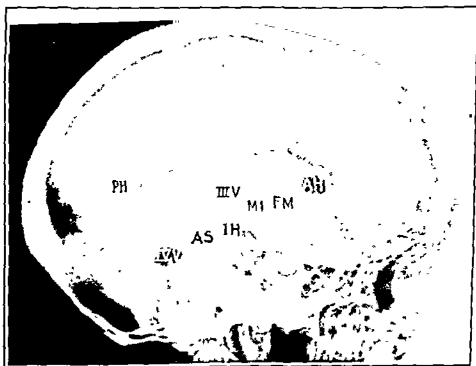


FIG 95 —Ventriculogram which shows normal ventricles filled with air The anterior horn (*AH*), posterior horn (*PH*), inferior horn (*IH*), third ventricle (*III V*), massa intermedia (*MI*), aqueduct of Sylvius (*AS*), fourth ventricle (*IV V*), and foramen of Monro (*FM*) are clearly shown.

injection. Therefore, he suggested the introduction of air into the spinal subarachnoid space so that as the air rose in the spinal canal it would then enter the basilar subarachnoid cisterns, the subarachnoid spaces over the cerebral hemispheres, and the ventricles. In such a manner it became possible to demonstrate any possible obstruction to the circulation or absorption of cerebrospinal fluid. This procedure he termed *encephalography*. (Figs. 94 and 95.)

It was, therefore, quite logical that Dandy should apply the knowledge gained from these studies to the difficult problems of diagnosis which are encountered in many patients with intracranial tumors. As is not uncommon, the use of these procedures immediately became widespread and startling claims were made for their diagnostic value, unexpected fatalities occurred and they were used in cases in which they were wholly unnecessary or directly contraindicated. The methods were employed without a knowledge of the anatomy and physiology of the cerebrospinal fluid pathway; interpretations were made without a personal knowledge of the details of the injection of the air; and encephalography was advocated as an office procedure.

Today, the pendulum has become stabilized so that a reasonably judicious viewpoint of the employment of these procedures has been reached. The frequency with which encephalography or ventriculography are used varies considerably in various clinics, depending upon the accuracy and detailed completeness of repeated neurological examinations. Of their diagnostic value, there can be no doubt and there is no question that improperly employed, both procedures are accompanied by danger. Encephalography is more dangerous and causes a more severe general reaction than does ventriculography, particularly in cases of increased intracranial pressure. In the presence of a papilledema, encephalography should never be employed in our opinion; nor should it be used if a posterior fossa lesion is suspected even though papilledema does not exist.

Various gases have been used as contrast media but air has been proved to be as satisfactory as any. Oxygen provides good films but the rapidity with which it is absorbed, makes it imperative to work more rapidly than when air is introduced. The discomfort, headaches and shock that accompany the introduction of air into the spinal canal constitute the most serious objection to encephalograms We have found, with others, that the inhalation of oxygen following the completion of the films relieves the majority of patients quite promptly and we have adopted it as a routine part of the procedure. In a considerable number of patients, it is impossible to

fill the ventricles satisfactorily by the injection of air into the spinal subarachnoid space. Just why this is true, particularly in patients without intracranial pathology, is not known. The subarachnoid spaces may be outlined in perfect detail, but the failure of the ventricles to fill makes it necessary to perform a ventriculogram.

The most difficult aspect of ventriculography or encephalography is the correct interpretation of the roentgenograms. In our opinion, it is essential that the individual who performs the injection of air should have a major rôle in their interpretation. He is the one who should have a grasp of the clinical signs and symptoms, however meager. He has obtained impressions and data, particularly in ventriculography, which are invaluable in forming a well-rounded opinion of the roentgenograms. It is also our opinion that both procedures are surgical operations, because it may be necessary in many instances to operate upon the patient immediately following the injection of air. In many clinics, air studies are done just prior to the craniotomy.

In a normal ventriculogram in which the ventricular system has been well filled with air, one learns to evaluate certain findings above others. To our mind the appearance of the air-filled ventricles shown on the roentgenogram taken with the occiput upon the film is extremely helpful and less likely to be misinterpreted by a failure to have introduced air in a sufficient amount to fill the ventricles completely.

Normally, the anterior horns are roughly triangular in shape and resemble butterfly wings. The superior and external sides of the ventricle represent the base and hypotenuse of a triangle. The medial edge of the triangle is a straight line which forms an obtuse angle with the base. The septum pellucidum separates the medial edge of each ventricular shadow which normally should be an equal distance from the midline. The third ventricle shadow is narrow and oval and lies inferior to the anterior horn shadows and in the median line. Very often a much lighter air shadow which is globular in shape lies lateral to the inferior half of the typical butterfly wing. This is the body of the ventricle which lies more lateral than do the poles of the anterior horns.

The lateral roentgenograms of the air-filled ventricles should be viewed steroscopically to obtain the greatest amount of information from their size, position and shape. Often the inferior horn may appear to be deformed when only a filling defect is present.

The roentgenograms taken with the forehead upon the film make it possible to visualize the bodies, inferior and posterior horns of the ventricles. The air shadows are horn-shaped and the convexity of the curve is away from the median line. The medial portion of the air-filled ventricle and the posterior horn are superimposed.

[illegible]

ge[illegible]

fo[illegible] a more complete description of the use of air in studying the ventricular system is "The Normal Encephalogram," Davidoff and Dyke—Lea & Febiger, 1937

A space-occupying lesion in the cerebral hemispheres will tend to collapse the homolateral ventricle and displace it toward the opposite side. The ventricle may be so collapsed that no air enters it. At the same time that the lateral ventricle is pushed to the opposite side, the third ventricle also becomes displaced beyond the median line. If the homolateral ventricle is partially or completely obstructed, the opposite ventricle will dilate. One of the earliest evidences of dilatation of a ventricle is the rounded appearance of the anterior horns. If there is an obstruction by a tumor in the third ventricle, aqueduct of Sylvius or posterior fossa, both ventricles dilate equally

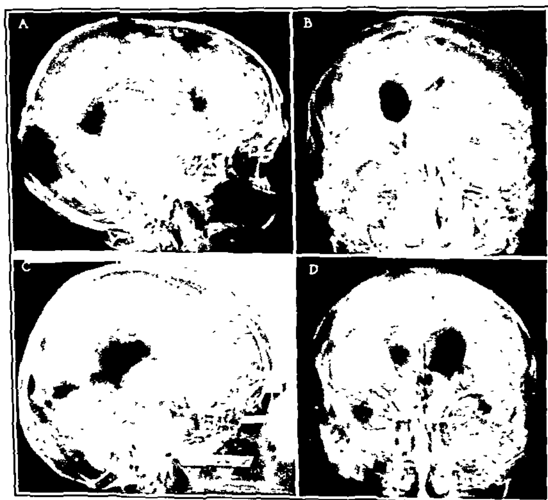


FIG 95 —Ventriculograms which show evidence of an intracranial space-occupying lesion: (*A*) lateral film showing absence of filling of the inferior horn; (*B*) anteroposterior view of the same patient as (*A*), which shows deformity of the left anterior horn, displacement of both ventricles to the right, and a marked shift of the third ventricle to the right; (*C*) lateral film showing a dilated lateral ventricle produced by a tumor of the posterior fossa; (*D*) anterior horns of the ventricles of same patient as (*C*), which show the symmetrical dilatation of the lateral ventricles and third ventricle produced by a tumor of the posterior fossa.

and a secondary hydrocephalus is produced although the inferior horns do not dilate as much as the anterior horns and body. (Fig. 96.)

Several years ago, Grant reported 392 cases of ventriculography from the members of the Society of Neurological Surgeons. Positive information as to the location of the tumor was obtained in roughly 80 per cent of those cases. In 30 per cent, definite facts as to the situation of the neoplasms were given when all other evidence was absent and nearly 80 per cent of the tumors localized by ventriculography in the absence of neurological evidence could be removed surgically. Because of misinterpretations, 1 per cent were operated upon incorrectly. The mortality rate was reported as 8 per cent, a figure which at the present time is far too high. The reduction of the mortality from this procedure has been due to a wiser choice of cases, a more accurate technique, and, finally, to a recognition of the fact that all preparations should be made for an immediate operation, if it suddenly becomes necessary.

Angiography.—Moniz first called attention to the diagnostic *possibilities of visualization of the cerebral vessels by the use of con-* trast media introduced into the carotid artery. Water soluble contrast substances of the type of diodrast have found an ever increasing use for cerebral angiography and have supplanted thorotrast. However, the injection of these media is followed by a sensation of pain and burning in the unanesthetized patient. They cause vasoconstriction and obviously have an irritating effect on the vascular walls.

Cerebral angiography is now a well established diagnostic procedure in many intracranial neurological problems. The contrast medium may be injected into the carotid artery percutaneously or by surgical exposure of the vessel. The former method is time saving but depends for its success upon the repeated experiences of the surgeon. In any event, angiography is not to be undertaken lightly or without thought of the maintenance of a perfect airway during the procedure.

A series of at least three *roentgen-ray exposures should be made* during the injection of the contrast medium to demonstrate the arteries and veins It must be appreciated that each exposure illustrates the cerebral vascular anatomy of that particular moment only and the state of the circulation in the cerebral vessels during most of the time of the injection can only be inferred. Curtis[1] has developed a method of obtaining rapid serial exposures which give a complete *description of the circulation of the medium* Twenty-five roentgen pictures may be obtained at intervals of ½, ⅓ of 1 second.

The use of rapid serial angiography has contributed greatly to the possibilities for physiological study of the cerebral circulation For

[1] Curtis, J. B. Rapid Serial Angiography, Preliminary Report, J Neurol, Neurosurg and Psychiat., 12, 167, 1949

example, it is not uncommon to see bilateral filling of the anterior cerebral arteries, so that the cerebral circulation is not so exclusively unilateral as has been supposed.

Angiography was used in the beginning as a method for the demonstration of intracranial vessel aneurysms and arteriovenous connections in angiomatous malformations. In this respect it has no equal and is indispensable in preparation for a surgical attack upon these lesions. The next development was its use to demonstrate the extent and pattern of the circulation of intracranial tumors. Evidence is accumulating which indicates that specific tumor types show characteristic vascular patterns.

Electro-Encephalography.—In 1935, the experiments of Berger on the electropotentials of the brain were applied clinically, and in the succeeding years the variety of normal and pathological conditions in which tracings of brain activity have been made has increased apparently without limit.

The value of the electro-encephalogram in clinical diagnosis must be based upon an accurate knowledge of the electrophysiology of the underlying brain. At present, the methods and techniques are being constantly improved so that a group of individuals well grounded in electrophysics devote themselves solely to this diagnostic procedure. The tracings are made by a specially built and sensitive apparatus and recorded in much the same way as an electrocardiogram, except that the poles are applied to the scalp.

Cerebral activity of any kind causes alterations in the normal waves and this forms the basis for a clinical application at present concerned with the localization of intracranial tumors, cortical scars and epileptogenous zones. There are three types of waves normally; the *alpha* waves are the most common and are most easily obtained from the occipital lobes; they have 10 oscillations per second. The *beta* waves are smaller, have a frequency of 20 to 25 oscillations per second and occur more prominently in the frontal area. The *gamma* waves range between 25 to 50 oscillations per second. *Delta* waves of slow frequency have been described as being special waves characteristic of grand mal and petit mal epilepsy. Electropotential waves are not influenced by general muscular action but are affected by the muscular action potentials of the scalp muscles.

The technical difficulties involved in making a tracing are many and in a beginner's hands, waves can be obtained from inert objects. Outside currents from radio and roentgen-ray equipment must be carefully screened out and other electrophysical factors, even more technical in nature, may be shown to play an important part in the tracings. Those who have been working in the field from the beginning are not yet in agreement as to which cell layer or layers of the

brain give rise to the waves. They do agree that a tracing consisting of *alpha* waves indicates primarily, automatic activity of the cerebral cortex; that *beta* waves represent cortical work and that *delta* waves indicate an interference with normal activity since they are found in deep sleep and in anoxia.

Scarff and Rahm[1] have made tracings directly from the exposed brain and have been unable to demonstrate sharp localization of spontaneous rhythms. In their tracings the *beta* waves predominate in frontal lobe areas but appeared also in the temporal and parietal lobes. *Alpha* waves were found in the parietal, temporal and occipital lobes.

The localization of neoplasms and epileptogenous scars by electroencephalography is now a matter of routine in many clinics. According to Case and Bucy,[2] tumors and some other lesions give rise to slow waves and sometimes spikes which arise from disturbed tissue around the tumor. Scarff and Rahm have found tumor tissue to be inactive electrically but have also recorded slow waves in tissue immediately around neoplasms. In the presence of benign growths (meningiomas) they have found abnormal electrical action to be circumscribed in the surrounding tissue but in gliomas, the majority of which are infiltrating in character, "there is a widespread change from slightly shifting alpha frequency downward on the edge of the invasive process to an absence of electrical activity in areas where degeneration has occurred."

The diagnostic value of this method depends understandably upon the ability to interpret the tracings. It has been our experience that it is not sufficiently accurate to be depended upon alone in the diagnosis of intracranial pathology.

Radioactive Tracer Dye Test.—In 1948, Moore[3] first used radioactive di-iodo-fluorescein in the diagnosis and localization of intracranial tumors. After a method was developed by Tabern for the production of stable di-iodo-fluorescein, its use was reported upon in 200 patients.[4] Experiences with this newer diagnostic procedure have multiplied rapidly and the apparatus, methods and techniques are being improved constantly, since the initial records of the accuracy of the method were so encouraging.

[1] Scarff, J. E., and Rahm, W. E. The Human Electrocorticogram, Trans. Am. Neurol. Assn., p. 20, 1940.

[2] Case, T. J., and Bucy, P. C. Localization of Cerebral Lesions by Electroencephalography. Jour. Neurophysiol., 1, 245, 1938.

[3] Moore, G. E. Use of Radioactive Di-iodo-fluorescein in Diagnosis and Localization of Brain Tumors, Science, 107, 569, 1948.

[4] Davis, L., Martin, J., Ashkenazy, M., LeRoy, G. V., and Fields, T. The Clinical Use of Radioactive Di-iodo-fluorescein in the Diagnosis and Localization of Tumors of the Central Nervous System. Jour. A.M.A., 144, 1424, 1950.

The test is based upon the known affinity of the radioactive dye for tumor tissue and specifically for the changed cellularity and vascularity of the tumor. The more malignant the neoplasm, the greater the radioactive fluorescein concentration. By the use of a Geiger-Mueller apparatus, counts are made over 32 fixed positions on the skull and these are recorded by tracings which can be read and differences in concentration of the dye noted. No untoward effects have been observed from the dosage of dye used which varies from 1.0 to 1.2 millicuries of radioactive material.

The apparatus used must be precisely calibrated and all phases of the test must be done with a standardized, accurate technique. It is also important that he who interprets the tracings be familiar with the various normal findings in all the 32 points of the head which are routinely studied. In the beginning, the test was time consuming and expensive but changes in technique and instrumentation have improved these conditions.

The test should not be repeated in doubtful cases until after five days have elapsed, to make certain that all radioactivity has disappeared from any possible focus of poor circulation where during the first test the material may have been pooled and from which it is slowly absorbed. The test is not reliable after acute head injury, after pneumoencephalography, or after a bout of convulsive seizures, because of the temporary disturbance of intracranial vascularity which such conditions are known to produce. The di-iodo-fluorescein test does not differentiate an acute infection from an anaplastic tumor, since the disturbed capillary permeability in both types of lesions will result in an increased exudation and pooling of the dye. But an acute inflammatory condition, if generalized, will give an all-over abnormally high isotope reading, whereas a tumor will show a localized point of concentration. Acute, circumscribed hematomas with their surrounding inflammatory changes will be localized by the isotope test, but old, chronic, quiescent hematomas usually will not thus be found. If a tumor is cystic, or if it contains a large, central, necrotic and gelatinous mass, it may be located by an abnormally *low* concentration of dye, as is also the case with an old abscess. In some tumors, as a metastatic nodule with an outer viable zone of tumor and a necrotic central cavity, the increased count produced by the active shell of tumor may be neutralized by the abnormally low count produced by the necrotic center, giving a count within the normal range. Errors have almost consistently been due to the presence of calcified, or non-vascular, or necrotic tumors, or tumors which are quite small and of moderate to low vascularity. The location of the tumor within the cranium is not a factor in errors. The accuracy of the test does seem to rely largely upon the presence of

abnormal capillaries in which the normal degree of permeability has been increased. As a rule, the more vascular the tumor the greater will be the concentration of the deposited dye.

Contrary to the use of angiography, and sometimes to the use of pneumoencephalography in either form, there have been no contraindications to the use of radioactive di-iodo-fluorescein under such conditions as an aged patient, advanced arteriosclerosis, recent thrombotic or hemorrhagic episodes, hypertension, or cardiac disease. Increased intracranial pressure, and tumors located in such dangerous places as the third ventricle, brain stem, and posterior cranial fossa, do not alter the safety of the use of the radiodye test.

A surgical Geiger-Mueller probe counter has been developed by Selverstone[1] which makes it possible to locate tumor tissue during an operation. Various refinements have been made in the construction of these instruments which make them more stable and useful. The identification of remaining tumor tissue after a radical removal of a glioblastoma, or other type of glioma, so that the surgeon may improve the attack upon these tumors is a significant contribution to the surgical treatment of intracranial tumors. Practically, the surgical probe counter has been proven and it remains only to improve the instrument and techniques.

When a neurosurgeon studies plain roentgenograms of a skull, pneumoencephalograms, arteriograms, electroencephalograms, or a radiodye graph, he has in mind certain preconceived notions concerning the patient's diagnosis, based on his knowledge of the history and findings upon physical examination It is a common experience for the neurosurgeon, with such knowledge at hand, to be able to detect small but significant changes in the results of the useful mechanical aids, not detected in routine examination by the roentgenologist or the technician who conducted the test Since his is the responsibility of the care of the patient, it should also be his responsibility to interpret in the light of his total knowledge of the patient, the results of all laboratory tests

RADIOTHERAPEUSIS IN INTRACRANIAL TUMORS

Though remarkable progress has been made in the surgical treatment of intracranial tumors, there are still many instances in which removal cannot be as complete as the surgeon would wish. This is particularly true of the gliomas It is true that the correlation of the pathological and clinical characteristics of this group of tumors has

[1] Selverstone, B., Solomon, A. K., and Sweet, W. H. Location of Brain Tumors by Means of Radioactive Phosphorus, Jour A M.A., 140, 277, 1949

made their subdivision possible so that we are now cognizant of the difference in malignancy between the astrocytomas on the one hand, and the glioblastomas on the other. However, the present surgical methods employed, which consist of removal of the tumor by suction, by resection of large portions of the hemisphere, or removal by the electrosurgical scalpel are not completely satisfactory. All of these methods entail a surgical risk, a gross destruction of brain tissue with the possibility of many neurological residual symptoms, and finally, the probability of leaving tumor tissue behind.

It was natural that the roentgen-ray and the rays of radium should be suggested in the treatment of intracranial neoplasms. In 1921, Ewing predicted that of all the intracranial tumors, the gliomas, which are made up of more embryonic cells, would react most favorably to roentgen-ray treatment. The experiences of neurological surgeons have substantiated this prophecy and in addition it has been learned that the more embryonic types of gliomas react most favorably.

Whereas, early apparently successful results with roentgen-ray therapy led some to advocate its use before surgery, it is at present agreed that surgery and roentgen-ray treatments should be used in conjunction and that no antagonism should exist between them. Without surgical verification one cannot be certain that a tumor is present. Nordentoft's experience is an example. In his series of 19 cases, there was only 1 verified neoplasm. The tumor in that particular instance was verified by an operation in which removal of the neoplasm cured the patient after prolonged roentgen-ray therapy had been carried out. Or, there may be a mistake in localization of the lesion and the maximum effect of therapy may not be received in the correct area. In the third place, several types of intracranial tumors are not only radioresistant, but offer favorable surgical results; for example, the meningiomas, the acoustic neurinomas and the astrocytomas. Finally, edema and increase of brain volume which follows roentgen-ray therapy may cause an acute increase in headaches and papilledema. These reaction symptoms may occur within one-half hour after radiation. Blood-vessels dilate, there is hyperemia and a serous transudate and edema occur. Numerous cases are upon record in which a single treatment of roentgen-ray has been followed by coma and death. Therefore, it must not be assumed that roentgen-ray therapy is devoid of danger.

The possible exception to the rule that surgical intervention should precede roentgen-ray therapy is the group of pituitary adenomas. Some believe that radiation is the treatment of choice in all cases of pituitary dysfunction irrespective of the presence of visual field changes or headaches. Anyone who has seen a sharply defined

bitemporal hemianopsia develop in a patient with pituitary dysfunction, in whom no previous visual symptoms existed, will realize that a qualification of that attitude is necessary. Certain it is that an hypophyseal adenoma should not be treated with roentgen-ray therapy without carefully taken visual fields made at frequent intervals. It is our custom to follow surgical removal by a course of roentgen-ray therapy and the technique employed has been given in the section dealing with the hypophyseal adenomas.

The gliomas offer the largest field for an addition to our therapeutic efforts by the employment of radiotherapeusis. In common with others, it has been our experience that the most embryonic type of tumor is affected to the greatest degree by deep roentgen-ray therapy. *We have made a microscopic comparison between sections removed* at operation and sections made later after roentgen-ray therapy had been given in 3 glioblastomas, 1 astrocytoma, 2 medulloblastomas and 1 ependymoma. In general the findings were that the tumor cells are damaged or, as in the case of medulloblastomas, finally disintegrate and there is a stimulation of connective tissue growth which seems to play an important part in retarding the growth of the tumor cells. The stimulating effect upon mesenchymal tissues in the medulloblastomas is very great. In our experience, there is no doubt that roentgen-ray therapy increases the average survival period of patients with medulloblastomas. Though the glioblastomas consist of poorly differentiated embryonic cells which might be supposed to be affected favorably by radiotherapeutics, we have yet to see such a case definitely benefited either by roentgen-ray or radium therapy. The astrocytomas and oligodendrogliomas are more radioresistant and their course is not materially lengthened by these methods. The wide extent and the enormous size and serpentine character of the angiomas usually force the surgeon to hold his hand and in this group of tumors radiation therapy offers distinct benefits which cannot be accomplished by surgery.

In the roentgen-ray treatment of gliomas, we use 220 kv. with 0.4 mm. tin, 0.25 mm. copper and 1 mm. aluminum filters with the tube at a distance of 20 cms. Four portals are used; frontal, 2 laterals, and occipital, which measure about 10 by 10 cms. in size. A total of 4500 to 6000 r units is given in any one series and the treatments are spaced in time according to the general reaction exhibited by the individual patient.

Developments in our knowledge of radium therapy have established certain fundamental principles that have an important bearing upon treatment. For example, it has become recognized that an optimum time interval exists during which a tumor reacts best to radiation. Exposure of the growth during a shorter period

results in failure to gain a maximum lethal effect upon the tumor cells. Exposure over a longer period results in the establishment of a state of radio-immunity. The importance of utilizing the most penetrating rays of radium has led to the use of adequate filtration, and a recognition of the significance of homogeneous distribution of radiation has resulted in the use of external radiation whenever possible and multiple implants when interstitial irradiation is indicated.

Since these principles have become recognized, it is not difficult to explain many of the failures of radium therapy under the older technique. The use of glass radon seeds, for example, invariably resulted in necrosis on account of the absence of filtration; and the frequent failures of gold implants can be explained on the basis of inadequate filtration and the difficulty of effecting a uniform distribution of radiation except in lesions of limited size.

The ideal form of radium therapy is that which permits the delivery of an adequate, uniform dose of penetrating irradiation over a prolonged, but limited interval, for example, by the use of a large quantity of radium at a distance. In the treatment of the more radioresistant tumors, however, the amount of radiation that can be delivered to the lesion by external radiation may be inadequate and interstitial radiation becomes necessary in order to deliver an adequate dose to the tumor.

Experiments now have shown that radioactive gold or phosphorus can be injected into cerebral tissue with minimal damage.[1] This encourages the injection of these materials into the tumor bed after a radical surgical extirpation of tumor tissue, so as to obtain direct radiation to the remaining neoplastic cells, or injection into a tumor which is identifiable but surgically unapproachable.

ARACHNOIDITIS

Many times acute or chronic inflammation of the arachnoid membrane results in an obstruction to the circulation of cerebrospinal fluid and localized and/or generalized symptoms are produced which are difficult to differentiate from space-occupying lesions. This is also true of thrombangiitis obliterans of the cerebral vessels.

The arachnoidea is a fragile membrane which covers the spinal cord and brain. Inflammatory lesions of this structure are of surgical interest because they may produce symptoms which are identical with those of an intracranial space-occupying lesion or a tumor of the spinal cord. Sometimes called "pseudotumors," "chronic serous

[1] Davis, L. and Goldstein, S.: "The Therapeutic Use of the Radioactive Isotopes in Intracranial Tumors," Ann. Surg., 136, 381, 1952.

arachnoiditis," "circumscribed," "adhesive," or "cystic arachnoiditis," these lesions have been encountered at autopsy or at operation in practically every part of the cerebrum, cerebellum and spinal cord.

The greatest portion of the literature deals with the localized or circumscribed process to which general attention was called by the first surgical case reported by Spiller, Martin and Musser in 1903. A spinal case was detailed in which all of the symptoms subsided after operation. Six years later, Horsley reported 21 cases of "chronic spinal meningitis," all of whom he had operated upon for the presupposed presence of a spinal cord tumor. The literature has become filled with similar reports but little has been said of the surgical aspects of the generalized form of arachnoiditis first described by Quincke under the term "meningitis serosa." Generalized arachnoiditis, which *produces intracranial symptoms more often*, is a distinct surgical entity which in our experience has produced several interesting, and baffling, clinical problems.[1]

We have encountered 194 of these cases with intracranial symptoms. In each instance we have verified pathological changes in the arachnoidea, but each patient has received a diagnosis of "tumor suspect" and persistent follow-up examinations are made in order that we may not overlook the possibility of a tumor by being lulled into a sense of false security by the diagnosis of arachnoiditis. This attitude has been adopted in spite of the fact that the symptoms have subsided in a high percentage of patients and have never recurred during several years. Such a critical attitude is necessary to avoid making this diagnosis too frequently at the operating table when a careful, patient, experienced examination of the brain, or meticulous search with the Geiger-Mueller surgical probe counter, would reveal the presence of a tumor. To date in only 5 cases of 194 in which arachnoiditis has been diagnosed at operation has an intracranial tumor been verified later

Our patients with intracranial symptoms may be divided into those who had predominating cerebral, cerebellar or optic chiasmal signs. 96 may be classed as cerebral, 44 as cerebellar and in 54 the presenting signs were rapid loss of vision with bizarre visual fields and severe headaches. Often these patients may have a rapidly progressing papilledema, headaches and no localizing or lateralizing signs. Many of the older patients in this group present marked mental symptoms in addition to their neurological signs.

A typical example of cerebral arachnoiditis, as we have encountered the condition, may be obtained from the following story. It

[1] Horrax reported 33 cases of chronic localized arachnoiditis with symptoms which pointed toward a process in the posterior fossa of the skull. Generalized Cisternal Arachnoiditis Simulating Cerebellar Tumors; Its Surgical Treatment and End-results. Arch. Surg., 9, 95, 1924.

must be emphasized, however, that headaches and a high grade papilledema alone may constitute the clinical picture for which surgery is urgently indicated.

The patient complained of severe headaches, visual difficulties, transient attacks of numbness, and inability to use the left side of the body. He had pneumonia in 1902 and influenza in 1918 and more recently in 1929 had had frequent attacks of arthritis. He had apparently recovered entirely from an operation on the nasal septum and right middle turbinate bone.

Early in the morning of July 7, 1929, he awakened with a terrific headache, definitely located on the right side. Ten days later he developed what he called "sinking spells," when he could not feel the bed and when he became very anxious and excited if left alone. A bilateral tinnitus soon developed and this was followed by numbness and inability to use the entire left side of his body, which lasted two days and disappeared with tingling sensations. These attacks were repeated and, during one, convulsive twitchings of the left side of the face occurred. At times he had the sensation of seeing loops of hair with the left eye and a tadpole which seemed to be definitely in the temporal field of the right eye.

There was a bilateral papilledema, a slight left facial weakness, a left-sided paresis with increase of the left deep tendon reflexes and diminished left abdominal reflexes. The Wassermann reactions on the blood and spinal fluid were negative

A right osteoplastic craniotomy was performed and a small nick into the dura and arachnoid was followed by a spurt of cerebrospinal fluid. There followed a large flow of fluid which reduced the tension so that the dura could be opened easily. The brain appeared as if it were enclosed in a gelatinous covering which resembled cellosilk. The surface vessels were enormously dilated and tortuous. The gyri were separated by deep sulci full of fluid and the arteries in the sulci moved in a serpentine fashion as they pulsated in the lake of fluid.

The patient made an uneventful recovery and resumed his work as a professor of political economy. In January of 1930 he had an attack of chickenpox during which he had a troublesome headache, peculiarly enough confined to the left side of his head for the most part. He made a good recovery from this illness without any intracranial symptoms.

In 4 young patients we have felt reasonably sure before operation, because of the localizing symptoms and the history of a recent otitis media, that an intracranial abscess was present but in no instance was an abscess found. In one child, left-sided symptoms subsided after exploration for an abscess and a right subtemporal decompression but almost immediately a right hemiparesis developed. This in turn disappeared following a left subtemporal decompression.

Forty-four patients have presented such definite cerebellar symptoms that a suboccipital craniotomy has been indicated. All of the cases have presented symptoms of increased intracranial pressure with [illegible] Cerebellar [illegible] Nystagmus, moderate dysmetria and incoördination in the upper extremities, ataxia and unsteadiness in walking, and symptoms referable to the

cranial nerves in the cerebello-pontile angle have been observed. The onset and course of the symptoms in this group of patients has varied from four months to two and one-half years before operation.

A third group of cases with supratentorial symptoms consists of 54 patients, whose symptoms were entirely confined to, or pointed predominantly to, the optic chiasm. We have come to look upon them as a definite clinical entity, closely related perhaps to a group of patients variously described by ophthalmologists as "retrobulbar neuritis." All of these patients complain of headaches and a progressive diminution of their visual fields while bizarre visual field defects and optic atrophy are the two most constant symptoms. Loss of visual acuity has always been described by the patient as a later symptom. In many cases the clinical symptoms are very difficult to differentiate from those produced by tumors which originate in the suprasellar region and in one young man, a diagnosis of Leber's disease, or heredo-familial optic atrophy, was made because of a familial history of blindness. In the remaining cases, the distribution between the earlier and later decades of life was equal and no definite etiological factors could be determined An illustrative case history is this one:

Except for an acute earache, the patient had been well until December of 1928 when she noticed that vision in the right eye was poor. By January 20, 1929 vision had failed in both eyes so that she was able only to distinguish between light and dark. She could recognize facial features in strong light but could not count fingers with either eye The right pupil was smaller than the left but neither reacted to light The discs were pale and sharply outlined and the retinal vessels were small.

At operation the optic nerves and chiasm could not be visualized until the chiasmal cisterns were emptied of fluid and the thick, gray, opaque arachnoid was dissected from the chiasm At the time of discharge the patient could count fingers readily, recognize features clearly, and could occasionally distinguish colors. She could read large news headlines and her visual acuity was 7/20 in the left eye and 2/100 in the right. Seventeen years later she was able to read 12 point type and out of doors was independent.

With but few exceptions, all of the patients were operated upon under the preoperative diagnosis of an intracranial tumor or abscess and in none was either of these conditions found. In every case a thickened, gray, opaque arachnoid membrane[1] was found over the cerebral hemisphere, cerebellum or about the optic chiasm and in the cerebello-pontile angles In many instances the brain has ap-

[1] Normally the arachnoidea is a fragile membrane which covers the brain and spinal cord and is made up of a thin, delicate, supporting reticulum covered on both sides by characteristic cells There are definite trabecular projections from the inner side of this membrane upon which the cells which form the lining of the membrane are continued The cells which cover the membranous expansion of the arachnoid are low and flat and have large, pale, oval nuclei with a very indistinct chromatin network.

peared to be enclosed in a dull cellophane bag. The arachnoid is so thick and tough that it can be grasped with tissue forceps and a section removed for microscopic study. When the arachnoid is opened, trapped cerebrospinal fluid under pressure spurts out and continues to escape freely in spite of reduction of cerebrospinal pressure by a ventricular puncture. We have been forced to dissect the arachnoid from the optic nerves and chiasm to free them after sucking out a lake of fluid which completely surrounds the chiasm, and commonly isolated small lakes of fluid may be drained by opening the thickened arachnoid in the cerebral sulci. It is true that one may often find the same kind of gray, opaque arachnoid over the cisterna magna when a cerebellar tumor is present or in the cerebello-pontile angle overlying an acoustic neurinoma, so that one should not make the diagnosis of arachnoiditis unless a careful examination fails to reveal a tumor.

Many of our patients gave a fairly recent story of an upper respiratory tract infection, or an otitis media with a significant rise in temperature but whether or not there is a direct relationship is difficult to say. We have been interested in such an etiological relationship but have never come to a decision as to whether or not these cases are analogous to the condition which Kennedy has described under the name of "acute benign meningo-encephalitis." Horrax has mentioned a case studied very carefully pathologically which showed a concomitant encephalitis, and 2 other cases showed sclerotic changes in the cerebral cortex as evidence of previous encephalitis. In one brain which we examined, there were no microscopic evidences of an encephalitis, but neither was there a history of any previous systemic infection.

We have made a pathological study of each section of arachnoid removed at operation and in 1931 Haven and Davis[1] reported their ability to differentiate inflammatory, fibrous and hyperplastic types of arachnoidal reaction The microscopic picture of the inflammatory type is characterized by a marked small round-cell infiltration into the arachnoid, a metamorphosis of some of the arachnoidal cells into gitter cells, a moderate amount of beginning fibrosis and a marked amount of inflammatory debris. In a second type, the predominating picture is a fibrosis which seems to involve only the reticular layer At times there is a moderate hyperplasia of the membranous cells of the arachnoid and occasionally a small round-cell nucelus is found in the fibrous reticulum. The largest number of cases in our series fall within this group. The hyperplastic arachnoidal reaction is characterized by a large number of arachnoidal

[1] Davis, L., and Haven, H.: A Clinico-pathological Study of the Intracranial Arachnoid Membrane, Jour Nerv. and Ment. Dis, 73, 129, 1931.

cells with a destruction of the normal architectonic structure of the membrane. The nuclei stain more darkly than normally and numerous mitotic figures are observed.

Of the 194 patients operated upon and in whom the diagnosis of arachnoiditis was verified by microscopic study of the membrane, all but 19 are alive from one to twenty-five years following their operation. In 5 of these 19 patients an intracranial tumor was verified at autopsy; in the remaining 14 patients who died of various systemic diseases an intracranial tumor was not found. This clinical and pathological condition in the brain and spinal cord is a distinct and definite entity but the diagnosis must be reserved for cases verified pathologically and by later clinical developments. In other words, it is a diagnosis which may easily be abused and like that of "chronic appendicitis" may cover a multitude of diagnostic sins and satisfy an undemanding surgical conscience.

CEREBRAL THROMBOANGIITIS OBLITERANS

Intracranial vascular lesions may simulate a tumor of the brain and frequently they are associated with disturbances in the peripheral vessels. There are, however, intracranial vascular disorders which are not associated with peripheral vascular disease and which create symptoms often impossible to differentiate from those of a space-occupying lesion. One of these is the cerebral form of thromboangiitis obliterans, also known as von Winiwarter-Buerger's disease

Until recently thromboangiitis obliterans was known as a disease affecting primarily the blood-vessels of the periphery of the extremities, particularly those of the toes and the fingers. Its clinical picture was characterized by paresthesia of the extremities, circulatory troubles of the cutaneous vessels, a feeling of rapid tiredness when walking, with attacks of pain producing intermittent claudication. At first these symptoms may appear to be functional in nature until one finds diminished or lost pulsation of the peripheral arteries and slowly progressing gangrene of the extremities From several clinical reports and particularly the anatomical examinations of autopsy material by Jaeger,[1] it is known that the cerebral vessels are often similarly affected. Thromboangiitis obliterans may start in the peripheral vessels and follow in the central vessels, or may involve peripheral and central vessels simultaneously, or it may start in the central vessels and either not involve the periphery at all, or affect it only later.

[1] Jaeger, E.: "Zur pathologischen Anatomie der Thromboangiitis obliterans bei juveniler Extremitaetaengangraen" Virchow's Arch. 284, 526, 1932.

This process has been seen to affect the vessels of the retina, the vessels of the kidneys, the stomach and other abdominal organs. It may affect the aorta and the coronary arteries producing infarctions in the heart musculature; it may affect the vessels of the endocardium, thus explaining many cases of sudden cardiac death in young people. In a study of 500 cases of peripheral thromboangiitis obliterans Hausner and Allen[1] found the evidence of a cerebral lesion in 11, or 2 per cent of the cases.

The cerebral form of the disease was first observed and described by Foerster and Guttman[2] and offers many diagnostic difficulties. Symptoms of increased intracranial pressure are rarely present and often the symptoms are only those of a localized cortical pathological process. Of 34 cases of true cerebral thromboangiitis obliterans reported in the literature prior to 1947, a diagnosis of cortical atrophy due to a vascular lesion was made in 13 cases following encephalography or ventriculography. The air studies in all of our own cases have shown a unilateral or bilateral internal hydrocephalus and enlarged subarachnoid spaces over one or both hemispheres.[3] The disease occurs most commonly between the ages of thirty and fifty years and in our series there have been 29 females and 13 males. The course of the disease is chronic and progressive, but frequently presents sudden attacks of temporary symptoms and complete remissions. The symptoms depend upon the location and the extension of the diseased vessels and for this reason they can be numerous and widespread. The most common are headaches, paresthesias, pareses, apraxia, asterognosis, hemiplegias, aphasia, dysarthria, and visual defects. Epileptiform seizures of the Jacksonian type occurred in 2 of our patients and psychic changes and memory defects are frequently present.

In none of our cases has the retina shown any of the changes which have been described as periphlebitis retinæ, and vascular spasms or papilledema have not been observed. The usual symptoms of peripheral vascular disease were present in only one of our patients upon whom the diagnosis was accurately made before verification by operation. In the remaining cases pulsations were normal in the radial, posterior tibial and dorsal pedis arteries. Plethysmograms taken of the fingers of one of the patients were normal, but showed no difference after the application of heat. The oscillometric read-

[1] Hausner, E., and Allen, E., "Cerebrovascular Complications in Thromboangiitis Obliterans," Ann. Int. Med., 12, 845, 1938.

[2] Foerster, O., and Guttman, L. "Cerebrale Komplikationem bei Thromboangitis obliterans" Arch. f. Psych at 100, 506 1933.

[3] Davis, L., and Perret, G. Cerebral Thrombo-Obliterans, Brit. Jour. Surg., 34, 308, 1947.

ings were within normal limits in both arms, although slightly lower on the left side and equally and slightly diminished in both legs.

A rather typical story of these patients is illustrated by the following:

A thirty-four year old female had a history of gradual onset of clumsiness and paresthesia of the left hand following a fall while skiing. This condition gradually disappeared after four weeks' duration. After an interval of freedom from symptoms for ten months a paralysis of the left leg suddenly developed followed by convulsive seizures of her entire left side, weakness, clumsiness and paresthesia of both left extremities.

The neurological examination revealed a slight left facial weakness, and paresis of the left upper extremity, most marked in the hand The paresis of the left leg had completely disappeared. There was a subjective hypesthesia over the left upper extremity, impairment of joint and vibratory sense in the fingers of the left hand, a marked loss of stereognosis in the left hand, as well as a marked clumsiness. Rapidly alternating movements, finger-to-nose and finger-to-finger tests were poorly done with the left hand. All deep reflexes were hyperactive on the left side

A diagnosis of a right parietal lobe lesion, posterior to the Rolandic area was made. At operation an extensive yellowish and atrophic area posterior to the central sulcus was found, through which ran cortical vessels which were obliterated and appeared like thick white threads. (Fig. 97.)

Upon operation all of the cases have presented a similar typical picture. The arachnoid membrane over the exposed area was thickened, and the subarachnoid space was greatly widened and contained an abnormally large amount of cerebrospinal fluid, which spurted out on opening the membrane. Disseminated cortical areas were found in various degrees and stages of yellowish-brown discoloration, softening and atrophy. The gyri appeared small and granular and the sulci enlarged. The pial vessels, arteries alone in some cases and arteries and veins in others, resembled thin, worm-like white strings and were completely obliterated for a distance of 1 to 2 centimeters. Proximal to these obliterative changes vessels of normal caliber are present and in some dark thrombosed masses may be seen. Proximal to these thrombi the vessels appear normal and contain blood. All of these changes have been seen in many of the distal branches of the middle cerebral artery scattered over the frontal, parietal and temporal lobes. The cortex supplied by these vessels contained numerous small areas of discoloration and softening while other neighboring areas had undergone granular atrophy. Normal appearing gyri adjacent to the softened areas, and supplied by potent sister branches of the obliterated arteries, showed a greatly increased number of fine cortical arterioles and venules It seemed as if this were a compensatory process for the lack of blood supply in the diseased areas.

Microscopically, the obliterated blood-vessels showed a proliferation of the endothelial cells of the intima of the vessel, which is normally composed of one single layer of smooth flat cells. This proliferation is made up of several layers of irregular large cells which grow into the lumen of the vessel and seem to be connected with the thrombus. The thrombus usually completely obliterated the lumen of the vessel and was fixed to its wall at the place of the

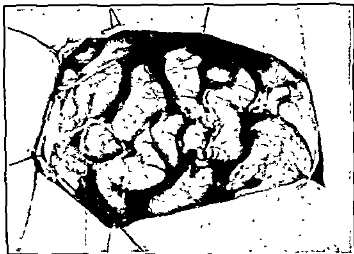


FIG. 97.—Appearance of brain of a patient with cerebral thromboangiitis obliterans. Note the obliterated vessels on the cortex which appear like thick white threads.

roughest endothelial proliferation. (Fig. 98.) It is organized and made up of a net of loosely connected cells, which contains pigment in its meshes, newly proliferated small vessels, some histiocytes and a few small cells which resemble lymphocytes. Leucocytic and lymphocytic infiltrations in the media and especially the adventitia of the vessels and also in the subarachnoid space surrounding them in the early stage of the disease have been observed but in later stages, like those we have encountered clinically, no fresh inflammatory reactions have been visible. A marked proliferation of the adventitia was present in each vessel. The underlying cortex showed multiple small areas of necrosis and glial proliferation at other places. This entire process may remain localized in one hemisphere or may be distributed symmetrically over both hemispheres in the boundary zone formed by the most distal branches of the anterior, middle and posterior cerebral arteries.

The etiology and pathogenesis of the cerebral form of thromboangiitis obliterans are the same as those of the peripheral form. Abuse of nicotine, trauma and non-specific infections have been held responsible for the disease; but this could only be possible on the

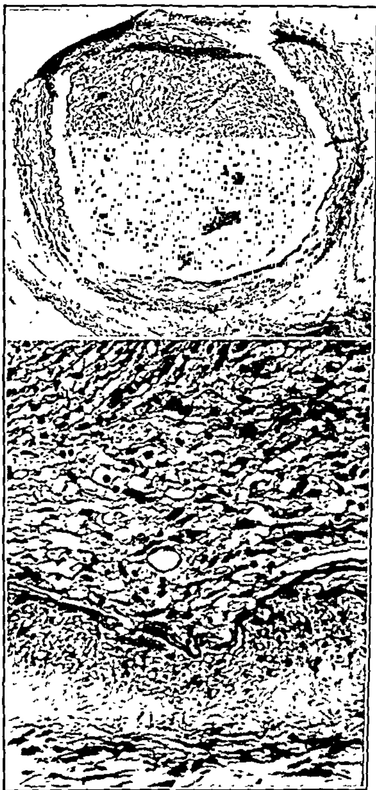


Fig. 98.—Obliterated vessels in cerebral thromboangiitis obliterans show proliferation of the endothelial cells of the intima. The thrombus is organized and made up of a net of loosely connected cells, some histiocytes and a few small cells which resemble lymphocytes It is fixed to the vessel wall at the point of the roughest endothelial proliferation.

basis of a pathological vascular constitution. The histological findings are not those of a bacterial or specific infection. It is not known whether the endothelial damage or the thrombus formation is the primary process, although it seems logical that the thrombus develops on the ground of the rugosity produced by the pathologic endothelial proliferation. It is possible that angiospasm such as those which have been observed in the retinal vessels may be the cause of the damage of the endothelial cells.

The anatomically proved changes cannot explain all the clinical disturbances. Many of the temporary symptoms must be produced by a temporary disturbance of a cerebral area which does not undergo later degeneration. Some of the symptoms do not depend entirely on the localization of the process but may be related to the degree of adaptability of the cerebral vessels and to the anatomic particularities of the supplied areas. A temporary decompensation of the collateral circulation, produced by variations of the blood pressure and the amount of circulating blood, and a sudden diminution of the blood supply to already poorly supplied areas may very well be the cause of the reversible cerebral damage and may be held responsible for many of the temporary symptoms.

The clinical syndrome of this disease entity becomes more familiar and, when considered with the rather characteristic air studies and the absence of concentration of radioactive dye, it becomes simpler to take a position against operation. We have had no experience with angiography in these patients but this may also be of help when correlated with the clinical signs and symptoms. One should always search diligently for evidence of peripheral manifestations of the disease.

CHAPTER IV

INFECTION OF THE CENTRAL NERVOUS SYSTEM

THE discovery of the antibiotics and sulfonamide drugs and the refinements in their use, but in particular the contributions which were made by Cairns and his colleagues[1] based on their experiences during World War II, have completely changed the prognosis of patients with infections of the central nervous system.

The high mortality rate in the treatment of meningitis and intracranial abscess led to the employment of heroic measures which were as varied in method as were the surgical specialists who treated them. There have been well defined eras in the treatment of intracranial abscess represented in the beginning by the otologist with his instrumental probings of the brain through an infected field. Then came the general surgeon who often ruptured the abscess wall with his exploring finger. As a result of experiences in World War I the Mosher brain drain was used, followed by neurosurgical attempts, at marsupialization of the abscess, closed drainage by a catheter, repeated evacuation by a brain cannula and finally, total excision of the abscess when a thick membranous wall has encapsulated it.[2] The complication of meningitis and ventriculitis made all of these methods hazardous until the antibiotics and sulfonamide drugs have made the occurrence of an intracranial abscess more and more rare and the treatment of meningitis more hopeful.

Intracranial Abscess.—Infection may reach the brain in any one of four ways: (1) by contiguous extension from an adjacent source of infection, such as the middle ear, mastoid or paranasal sinuses; (2) by direct implantation from a penetrating wound; (3)

[1] Cairns, H., Lewin, W. S., Duthie, E. S., Smith, H. V.: Pneumococcal Meningitis Treated with Penicillin, The Lancet, May 20, 655, 1944

Shiller, F., Cairns, H., Russell, D. S.: The Treatment of Purulent Pachymeningitis and Subdural Suppuration with Special Reference to Penicillin, Jour. Neur. Neurosurg. and Psych., 11, 143, 1948

Cairns, H., and Schiller, F.: Purulent Pachymeningitis, Proc. Royal Soc. Med., 41, 805, 1948.

Cairns, H., and Taylor, M.: A Review of the Treatment of Tuberculous Meningitis with Streptomycin, Proc. Royal Soc. Med., 42, 7, 1949.

Cairns, H.: Surgical Aspects of Meningitis, Brit. Med. Jour., 1, 969, 1949

Cairns, H.: Penicillin in Head and Spinal Wounds, Brit. Jour. Surg., 32, 199, 1944

[2] Fincher, E. F.: Craniotomy and Total Dissection as a Method in the Treatment of Abscess of the Brain, Ann. Surg. 123, 789, 1946

by metastatic extension through the blood stream and (4) from unknown sources of infection by pathways undemonstrable.

From a contiguous area of infection, extension to the brain may occur along the blood-vessels, or Virchow-Robin spaces, or through preformed paths, nerve sheaths, or foramina. In our own series, this is the most common type of extension, and infection of the middle ear, mastoid and frontal sinus are the principal foci. If the tegmen tympani or antri, or the zygomatic cells of the mastoid bone are the seat of the osteomyelitis, the majority of abscesses occur in the temporosphenoidal lobe of the brain which is in the immediate neighborhood of the petrous bone. However, frontal lobe, parietal lobe or cerebellar abscesses do occur from this source of infection.

From an adjacent focus of osteomyelitis, the method of spread of the infection to the brain is an important matter histopathologically and surgically. If the organisms are virulent, when the dura mater and arachnoid are reached and penetrated, meningitis will develop before an abscess of the brain forms, unless the subarachnoid space has been obliterated by adhesions between the dura, arachnoid and pia mater. Having reached the brain, the infection may then spread along the perivascular spaces or by retrograde thrombosis of the arteries and veins. Considerable controversy exists over the exact mechanism of this extension through the brain, although it is probable that a combination of these routes is the correct explanation. It is true that a retrograde venous thrombosis is the cause of many cerebellar abscesses, particularly when the lateral venous sinus is infected. Bagley[1] has shown that the superior petrosal sinus receives veins which empty from the tympanic cavity through adjacent dural veins, and from the cortex of the temporal lobe. Infection can therefore be transmitted from one area to the other with the formation of an abscess at a considerable distance from the original focus. Courville and Nielsen[2] and others believe that this is the manner in which infection spreads from the ear to the frontal lobe. This mechanism may also explain the method of spread from a frontal sinus infection into the brain. For example infection of the mucous membrane lining the sinus eventually reaches the bone; from the diploë of the bone, venous channels extend backward to the internal table and join with veins which penetrate the dura mater and join the cerebral vessels. When the veins of the dura mater are involved, infectious granulations cut off the blood supply to the adjacent bone of the skull and the osteomyelitis spreads. At some point infection may

[1] Bagley, C. Brain Abscess. Surg., Gynec. and Obst., 38, 1, 1924.

[2] Courville, C. B., and Nielsen, J. M. Fatal Complications of Otitis Media, With Particular Reference to Intracranial Lesion in Series of 10,000 Autopsies, Arch Otolaryngol., 19, 451, 1934.

pass through the dura and a focus of infection begins in the brain which eventually leads to an abscess. If adhesions form across the subarachnoid space, meningitis does not occur. As has been pointed out by Carmichael, Kernohan and Adson,[1] perivascular infiltration is present about every intracranial abscess and extension from infection of the subarachnoid space may be carried into the cortex along the perivascular spaces of perforating vessels.

Unless the dura mater is penetrated, an intracranial abscess rarely follows an injury to the skull but the injury to the dura mater may be very small and go unnoticed. Indriven spicules of bone, a bullet, shell or bomb fragments may be the primary focus about which an abscess develops years later. It is important, therefore, to emphasize that the immediate treatment of compounded injuries of the skull and brain should be thorough and painstaking to avoid such a late complication. That the original injury may be overlooked, or minimized, is illustrated by the story of one of our patients:

The patient had been attacked in a hold-up and was struck over the head by what he said was a milk bottle. The scalp laceration was only 2 inches long and had healed by primary intention. Three months later, he gradually developed a hemiparesis and complained of blurring of vision. Intermittently, he was stuporous and quite alert. He was operated upon under the diagnosis of a subdural hematoma, but in the course of the operation a fine linear fracture of the skull was seen and as the flap was raised, the point of a knife blade was found penetrating the dura. Beneath was an enormous encapsulated abscess.

Intrapulmonary infection, in our experience, is the most common cause of metastatic intracranial abscesses Lung abscess, chronic bronchiectasis, and chronic empyema are the most common primary foci. Osteomyelitis of other bones than the skull, or any local focus of infection in any portion of the body, may be the source of an intracranial abscess. Although reports in the literature state that such abscesses are more often single than multiple, the opposite has been our experience.

Yaskin, Grant and Groff[2] have reported 4 cases in which an intracranial abscess developed and in spite of a careful search no primary source of infection was discovered and the pathway of the infection was undetermined.

The development of an intracranial abscess is the same, regardless of whether infection reaches the brain along the perivascular spaces by retrograde venous thrombosis, or as a septic embolus. The rapidity with which it forms depends upon the virulence of the

[1] Carmichael, F. A., Kernohan, J. W., and Adson, A. W.: Histopathogenesis of Cerebral Abscess, Arch. Neurol. and Psychiat., 42, 1001, 1939

[2] Yaskin, J. C., Grant, F. C. and Groff, R. A. Brain Abscess of Undetermined Etiology; Report of 4 Cases With Recovery, Ann. Surg., 107, 492, 1938

organism and the resistance of the patient, just as is true of infection elsewhere in the body.

Globus and Horn[1] have described the early histopathology very accurately: "The earliest appearance of an abscess of the brain is that of a small circumscribed area crowded with bacteria, numerous polymorphonuclear leucocytes, lymphocytes, red blood cells, and disintegrating nerve elements."

For descriptive purposes, Carmichael, Kernohan and Adson divide the development of an intracranial abscess into four stages: (1) focal necrosis (microgliosis); (2) primary delimination (fibrosis); (3) secondary delimitation (astrogliosis); and (4) repair (vascularization).

The outstanding feature of the first stage is the central area of necrosis which is composed of polymorphonuclear cells, lymphocytes, monocytes and gitter cells. The blood-vessels are hyperemic; perivascular infiltration is present and there is an occlusive endarteritis, endophlebitis and hemorrhage. Microglia cells are the earliest elements to take part in this inflammatory reaction. Globus and Horn say that at this stage numerous adjacent blood-vessels become involved and as the necrosis from interference with the blood supply spreads, separate areas of necrosis coalesce to form the body of the abscess. At this time, the abscess consists of two layers; a central necrotic core and an ill-defined surrounding layer of hyperemia and microgliosis.

In the second stage of pathological change, the central area of necrosis is present unchanged but there is less evidence of acute infection. The microglia assume irregular-shaped forms or become rod-shaped Astrocytes appear in large numbers, but the outstanding characteristic is the appearance of fibroblasts in the zone of hyperemia immediately adjacent to the necrotic center. One group of neuropathologists believe that these fibroblasts arise from proliferating blood-vessels; another, that they are derived from lymphocytes which have migrated into the area of infection. Three layers are found in the abscess at this stage; the central area of necrosis which is liquefying, an adjacent region of hyperemia and fibroblastic reaction, and an external, poorly defined layer of astrogliosis.

A definite proliferation of blood-vessels marks the third stage of abscess formation. The new vessels have no perivascular spaces which indicates that they are newly formed. The microglia, astroglia and fibroblasts increase in number and the fibrous zone about the abscess center is thicker and more compact. Finally, the abscess

[1] Globus, J. H., and Horn, W. L.: Inherent Healing Properties of Abscess of Brain, Clinico-anatomic Survey of 15 Verified Cases, Arch. Otolaryngol., 16. 603, 1932.

to warrant. Kennedy has put it succinctly in stating that an abscess produces a "muddied intellect" to a greater degree than does any *other space-occupying lesion.* But the change from that state to relative alertness within a short period of time, as is so characteristic of a chronic subdural hematoma, may be striking.

Nausea and vomiting are no more common in the presence of an intracranial abscess than they are with any other space-occupying lesion which affects the intracranial pressure. A metastatic abscess *may produce a rise in the temperature,* but usually it is normal or subnormal. Slowing of the pulse and respirations, in our patients, have been much more common than in intracranial tumors. The *leucocyte count is not raised materially and certainly not significantly,* particularly if a sinus infection or a thrombosed dural sinus is present.

The optic nerve head changes which accompany an intracranial *abscess progress slowly and are quite characteristic in our opinion.* The disc becomes swollen and elevated, but in addition there are minute hemorrhagic streaks in the retina which radiate from the *nerve head. These surround the disc in such a way as to resemble* closely the commonly seen drawings of the radiations from the sun We have come to describe the appearance for ourselves as a "sunburst" appearance of the disc. The hemorrhagic areas in the retina often seen with a papilledema due to an intracranial tumor are quite different. Abscesses, like tumors, may advance rapidly and destroy cerebral tissue without increasing intracranial pressure, but certainly we believe that swelling of the optic disc occurs just as frequently in the presence of an abscess as a tumor.

There are many times when an examination of the spinal fluid aids materially in the diagnosis and decision for or against immediate surgical therapy. A sterile fluid, a predominance of lymphocytes in the cell count and a rise in intraspinal pressure speak for an abscess.

The symptoms just described above are all those of a space-occupying lesion but are not characteristic of an intracranial abscess. The latter diagnosis may be presumed if there is a definite history of an antecedent infection. However, in any event, they give *no* indication of the location of the lesion within the brain. The symptoms of localization differ in no manner from those described in the chapters on diagnosis and intracranial tumors.

The signs of localization depend upon the size and situation of the abscess. A frontal lobe abscess may reach an enormous size before symptoms are produced.

This was true in the case of a young man who was operated upon He had a right frontal sinusitis which had been inadequately drained He developed headaches and had *periods of dullness and drowsiness.* A more radical operation was performed on the sinus and his condition became so much improved

that he was sent home to report for examination each week. During the third week his headaches recurred and the optic discs were edematous. He was checked carefully for other symptoms, but none were found. Within ten days there was a slight weakness of the left side of his face. Based on this single localizing finding, an operation was performed and a large, encapsulated abscess was drained from the right frontal lobe. He has returned to his school work and is free from all symptoms.

This slight weakness of the opposite side of the face may often be the first neurological sign of any value. Gradually, the arm on the same side may become weak and the deep tendon reflexes increased. The detection of the small signs of motor involvement is particularly important in helping to localize an abscess.

Quite naturally, osteomyelitis of the frontal bone, or a sinusitis, suggests the lateralization of an abscess but definite neurological symptoms are more accurate and reliable. An abscess of the temporal lobe is most frequently encountered secondary to a middle ear infection but such an abscess may produce an homonymous hemianopsia, palsy of the third, fourth and sixth cranial nerves, contralateral paresis of the facial muscles, difficulty in naming objects or in using words and changes in the reflexes which are more dependable.

An abscess of the cerebellar hemisphere gradually produces the usual symptoms of cerebellar dysfunction. In addition, we have been impressed by the preference of the patient to lie on the side of the lesion and to resist any movement of the head to the opposite side because vomiting, vertigo and an increase in nystagmus are produced. We have never been able to detect any difference in the nystagmus produced by an abscess or a tumor of the cerebellum, though certainly the coarse, ataxic nystagmus produced by both of these lesions differs markedly from that of a labyrinthine nystagmus. Tenderness or rigidity of the neck are commonly present as they are in other posterior fossa lesions.

We have never received any help from roentgen-ray films of the skull except to determine the extent of an osteomyelitic process. We have never used ventriculography or encephalography to aid in the localization of an abscess, although it is recommended and practiced by many neurological surgeons. The use of contrast media injected into the verified cavity of an encapsulated abscess is helpful in following shrinkage of the size of the abscess cavity and often in outlining an outpocketing which may require drainage.

The surgeon must decide correctly where the abscess is situated; when he should evacuate the abscess and finally, how he should do it to avoid spreading the infection and at the same time completely drain the cavity.

Sir William Macewen first opened a temporal lobe abscess by trephining through a clean surgical field in the temporal bone and

later stated that he "now regards an uncomplicated cerebral abscess, early recognized, accurately localized, and promptly operated on, as one of the most satisfactory of intracranial lesions, the patient at once being relieved from a perilous condition and usually restored to sound health." There is no doubt that Sir William's optimism and high percentage of recoveries was due to the fact that the majority of his operations were delayed, either because of his good surgical judgment or by circumstances, until encapsulation had taken place. Following this pioneer work, Macewen's teachings were disregarded by those who were operating upon these cases, until neurological surgery became well established as a surgical specialty.

At present the surgical methods employed in the treatment of an intracranial abscess may be divided roughly into two types, depending upon the size of the opening made in the skull for drainage of the abscess. Like other neurological surgeons we have passed through several stages of developing an operative technique to deal with abscesses and at present we vary our procedure to fit the particular case as well as we can. We adhere to the principle of disturbing the brain and its surrounding coverings as little as possible. Small bone openings and attempts to seal off the tract from the surrounding brain and meninges has been a fundmental principle of each method of drainage we have used. Some surgeons believe that a small burr hole opening in the skull, evacuating the pus with a brain cannula, and the introduction of a small, soft rubber catheter as a drain will give excellent results in the majority of cases. We have never been convinced that the drain worked efficiently and for some time, we have confined our efforts to drainage by repeated introductions of the cannula into the abscess cavity. A rubber tube is introduced only with the idea that it will form a tract through which subsequently the cannula may be introduced, without passing through another layer of cortical tissue. This method has many advantages in small abscesses whose contents are relatively sterile but larger cavities continue to refill and there is danger of spreading the infection through the brain tissue by continued introduction and withdrawal of the cannula.

Another group of surgeons use a large bony opening and, we have also done this, seal off the meninges to the cortex, incise or excise the cortex down to the abscess wall, evacuate the pus under direct vision, pack the cavity with gauze and drains or, fix and shrink the cavity with Zenker's solution and coagulation with the electrosurgical unit. Others have left the cavity open and allow it to herniate and extrude itself.

When the abscess is drained with little or no destruction of the cortex, as in the first method described, the sequelæ of neurological

symptoms may be reduced but there is less opportunity to drain the abscess perfectly and the reduction of intracranial pressure is slower. Adequate and immediate drainage and relief of pressure are the result of the second method but it entails destruction of the overlying cortex which may give residual symptoms. We have had success with both methods and used the second in three instances in which we could elicit no history of an antecedent infection and believed that we were dealing with an intracranial tumor. After the osteoplastic craniotomy had been performed and the bone flap elevated, a thick, encapsulated abscess was discovered. In each case the overlying cortex was incised, the capsule wall exposed, coagulated, opened, drained and treated with Zenker's solution and the coagulating current until the abscess cavity had shrunken and was obliterated. In each case, the convalescence was prolonged by infection of the wound which in each case was successfully treated.

On the other hand, in another patient, a young girl who developed a frontal lobe abscess following a frontal sinusitis, repeated evacuation with a brain cannula through a small burr hole opening was the method employed successfully. It was necessary, however, to perform a subtemporal decompression on the right side (the presumed abscess was on the left), in order to control her rising intracranial pressure until we were more confident that sufficient time had elapsed for the abscess to have formed a good thick capsule.

The second method employed by a number of neurological surgeons consists in identifying the capsule of the abscess with an exploring brain cannula, then enlarging the incision in the scalp and widening the burr hole opening. The dura mater is then opened with a stellate-shaped incision so that the dural and bony openings are about equal. The subarachnoid space is then sealed off by packing iodine-soaked gauze between the dura mater and arachnoid, by suturing the dura to the cortex or by coagulating the dura, arachnoid and pia to the cortex with the electrosurgical unit; the method we believe to be the most efficient. The cortex is then incised down to the abscess wall, or is resected with the electrosurgical unit until the surface of the abscess cavity is well exposed. The [illegible]

[illegible]

tion as much as possible, the abscess wall is opened. The cavity is then evacuated under direct vision by suction. From this point on the procedure varies greatly and seems to us to be a matter of individual choice of the surgeon. Some men pack the abscess cavity with gauze, or rubber tube drains or both, and then begin to remove the packs after the first week. After about two or three weeks the drains have been extruded spontaneously by closure of the cavity.

Others pull the capsule upward into the defect in the bone, suture it to the galea or pericranium and thereby marsupialize the abscess, using the capsule to protect the cortex and subarachnoid space. Another surgeon follows the shrinkage of the abscess cavity and its extrusion by introducing thorotrast after the pus has been evacuated. As we have said before, in the past we have used Zenker's solution to shrink the capsule and then have coagulated the abscess wall leaving it in place, without disturbing its outer limiting boundary with the uninvolved cortex. Some surgeons are greatly concerned over removal of the capsule so that a large fibrous scar may not form subsequently and produce epileptiform seizures, but in our experience, a spreading encephalitis is likely to develop from an attempt to remove the capsule. A glial scar forms even though the capsule be removed and it may produce epileptiform seizures as well as the capsule of an abscess. Which patients may later develop convulsions after an abscess is drained is just as unpredictable as which ones will have seizures after injuries to the skull and brain. In our own series of 95 patients with intracranial abscesses, of those who have recovered from the lesion, 7 have epileptiform seizures which are controlled successfully by anticonvulsant therapy but each of these patients had seizures produced by their lesion before they were operated upon.

Prior to the use of penicillin and the sulfonamide drugs we had never seen an intracranial abscess recover which was considered to be an acute surgical emergency. In other words, if delay was not tolerated by the patient to permit encapsulation to occur, the results of surgical intervention were bad. Often a great deal could be gained by reducing intracranial pressure by a subtemporal decompression in those patients who appeared to be in serious danger and for whom immediate drainage seemed the only hope. Now, the systemic administration of the sulfonamide drugs and/or penicillin should be started the moment one suspects the presence of an intracranial infection

It is easy to establish and maintain an adequate level of the sulfonamides in the cerebrospinal fluid by oral or intravenous administration and, therefore, this step may well prevent or check the development of meningitis during the treatment of the abscess. To obtain the same result with penicillin it is necessary to inject the drug into the cerebrospinal pathways under strict aseptic precautions at not less than twenty-four hour intervals, with unfailing regularity Therefore, though often the sulfonamides are not as effective as penicillin in the treatment of intracranial infection, as has been demonstrated for example in the treatment of pneumococcic meningitis,[1] the ease with which they can be administered and with

[1] Cairns, H., Duthie, E. S., Lewin, W. S., and Smith, H. V. "Pneumococcal Meningitis Treated With Penicillin." The Lancet, May 20 p. 655, 1944

which they pass through the barrier between the blood and cerebrospinal fluid makes them extremely valuable as chemotherapeutic adjuncts in the surgical treatment of intracranial abscesses. It must be pointed out that chemotherapeutic agents given systemically have benefited experimental cerebral abscesses only in so far as associated meningitis, if present, was benefited In other words, experiments indicate that these drugs probably do not gain access to well-encapsulated infections by way of the blood stream, hence the advisability of injecting them locally into the abscess cavity.

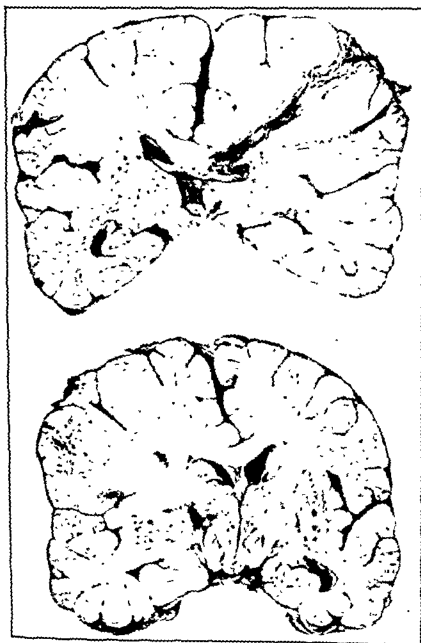


FIG 100 —Coronal sections of the brain of a patient which show one large abscess cavity which collapsed after drainage, and multiple small abscesses scatt through both cerebral hemispheres

A great deal of experience has been gained during World War II in the treatment of intracranial infections by chemotherapeutic and antibiotic agents. Penicillin was first used in recent penetrating cranio-cerebral wounds during the battle of Sicily by British neurosurgical units. As Cairns and his co-workers pointed out, to combat infection in wounds of the brain, penicillin must be administered *locally*, and they consider the drug to be innocuous both within the

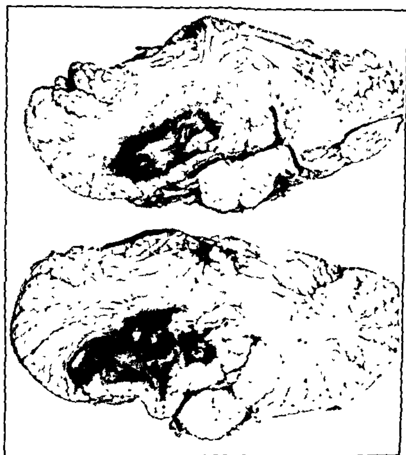


Fig. 101 —Coronal sections of the cerebellum which show an abscess without a limiting capsule.

brain substance and in the cerebrospinal pathways in concentrations well above those required to inhibit the growth of sensitive organisms.

Neither the administration of the sulfonamides or penicillin will prevent the development of an intracranial abscess following a compounded cranio-cerebral injury if the wound is not thoroughly treated surgically. Retained fragments of bone are far more frequently the nidus about which an abscess develops than are metallic foreign bodies A suppurating sinus infection may be the original

focus of an intracranial abscess or a meningitis and must be treated surgically to accomplish a successful outcome even though the pyogenic meningitis can be satisfactorily overcome by regular daily intrathecal injections of penicillin. (Fig. 100.)

In our experience, cerebellar abscesses are usually small, deep-seated and have a much thinner capsule than cerebral abscesses. They are better treated by small burr hole openings in the bone and drainage by repeated evacuation with a brain cannula. (Fig. 101.)

We have in our series 6 patients, all of whom but one have been children, who have been classified as cases of "pseudo-abscess" or "arachnoiditis." Just exactly what the histopathological condition is as yet we do not know. In each instance there has been a definite history of otitis media and mastoiditis which have preceded the slowly progressive symptoms of cerebral involvement. In all of these cases definite localizing symptoms have been present and an operation has been performed without finding any evidence of an abscess. In every instance, however, the arachnoid membrane has been greatly thickened, and beneath it a tremendous amount of cerebrospinal fluid has collected. In each case, we have enlarged the craniotomy to the size of a subtemporal decompression opening and have opened the dura mater widely. In one child, this train of symptoms occurred later on the opposite side again following a mastoid infection. Again, the same procedure was carried out in the belief that surely an abscess would be found. All of these patients are well and all of their symptoms have disappeared, though in every case the prominent pulsating decompression opening has been slow in receding. The long periods of freedom from symptoms which have followed operation in these patients make it seem unlikely that an abscess was overlooked in each case. Perhaps, the diagnosis of an osteomyelitis of the petrous bone in these patients would be more correct.

Intracranial abscesses are most difficult to treat and it requires the most accurate and astute surgical judgment to operate upon them successfully. If the virulence of the organisms and the natural defenses of the body are such that encapsulation does not occur, any surgical procedure is tremendously handicapped. Antibiotics and the sulfonamide drugs should be used intensively to aid the patient in the period of watchful waiting. Encapsulation of the abscess makes it possible for the surgeon to drain it or totally excise it successfully.

Meningitis.—Obviously, the more adequate surgical care of infections of the middle ear and mastoid, and improved methods of surgical cleansing and suturing scalp wounds has reduced the incidence of meningitis. It is still important, however, to remember that the release of pus by incising the ear drum or the time required to

cleanse and suture a wound meticuously are valuable prophylactic steps.

The mutiplicity of surgical methods for treating meningitis are as numerous as those concerned with the treatment of intracranial abscess. All of the surgical methods used were designed to accomplish the application of antiseptics and antisera, the continuous or intermittent drainage of cerebrospinal fluid or, the irrigation of the subarachnoid spaces and ventricles with physiological fluids. Continuous drainage of the cerebrospinal fluid from the cisterna magna, the lateral ventricles, or the lumbar subarachnoid space was performed by many in an effort to reduce intracranial pressure, drain the inflammatory products, promote an increased flow of cerebrospinal fluid and thus dilute the toxins. Large doses of hypotonic fluids were given parenterally and orally to promote the flow of cerebrospinal fluid. However, none of these surgical methods were particularly helpful and they were all relegated to a subsidiary place, when the sulfonamide drugs showed their value. After 1942, following the discovery of penicillin and other antibiotics, surgical methods became necessary for the introduction of these drugs because penicillin, for example, does not pass the blood-cerebrospinal fluid barrier in sufficient amounts to be effective.

Relatively, the brain is resistant to bacterial infection whereas the ventricles and basilar subarachnoid spaces are highly susceptible. In addition to the usual pyogenic organisms which produce meningitis, bacteria which are supposedly saprophytic to man, such as Micrococcus tetragenous, may produce an adhesive type of meningitis which blocks the cerebrospinal pathways completely.

In the acute stage of meningitis, the intracranial pressure rises but is not a serious factor unless a block in the cerebrospinal pathway exists. Often pus, fibrin or granulation tissue may obstruct the foramina of Monro, the acqueduct of Sylvius, the foramina of Magendie and Luschka, the basilar subarachnoid cisterns and the spinal canal. Thus, cerebrospinal fluid may not escape from the ventricular system and so there is little or none in the lumbar subarachnoid space Or, it escapes from the ventricular system, distends the basilar cisterns and spinal subarachnoid space, but does not reach the cerebral subarachnoid space where it can be absorbed. In this instance, antibiotics injected into the ventricles reach the spinal subarachnoid space very easily. However, obstruction in the spinal subarachnoid space prevents antibiotics administered by the lumbar route from reaching the ventricles and basilar cisterns.

One of the discouraging features of the treatment of meningitis in the past has been the disappointing relapses of the condition when the patient has seemed on the road to recovery. This still occurs

though many varieties of meningitis have been brought under control by the sulfonamides and antibiotics. Infection from the petrous bone and overlying dura, through fracture lines involving the paranasal sinuses, or from minute abscesses or collections of fibrinopurulent material in the subarachnoid spaces may produce relapses or recurrences of meningitis. The antibiotics do not penetrate these non-vascular areas of infection to any great extent and therefore a proper concentration of the appropriate antibiotic must be maintained in the cerebrospinal fluid until the fibrinous area is absorbed with release of all the enclosed bacteria or until dense fibrous tissue encloses the area, as in tuberculous meningitis.

The role of surgery in the treatment of meningitis is clear and the indications are definite. Meningitis can be prevented by the proper meticulous cleansing and suturing of a scalp wound. An individual with a fracture of the anterior fossa of the skull with an unclosed dural tear over a broken ethmoidal or frontal sinus and patients with a persistent cerebrospinal rhinorrhea or aerocele after head injury should all be suspect of developing a meningitis. Ventricular estimation and ventriculography are often important in the early diagnosis of meningitis which is a matter of great importance now that effective methods of treatment are possible. Often, bacteriological examination of the ventricular fluid will be fruitful when repeated examinations of the lumbar spinal fluid have failed.

The introduction of penicillin directly into the ventricles in cases of pneumococcic meningitis is reserved for those patients in whom there is a block in the spinal subarachnoid space. Cairns and his colleagues have shown that penicillin injected into the unobstructed lumbar subarachnoid space will spread quickly into the ventricles and cerebral subarachnoid spaces. From experiments which they carried out before the antibiotics were discovered, Pollock and Davis are inclined to believe that the spread of these substances from the lumbar spaces to the ventricles or cerebral subarachnoid spaces would be facilitated and insured, if frontal burr holes were placed in the skull and the dura mater was incised. Often the antibiotics may be introduced into the cisterna magna but severe retraction of the head or restlessness may make this a dangerous procedure. It may be necessary to introduce penicillin directly into the subdural space, where free pus may be circulating in purulent pachymeningitis, by means of an indwelling catheter. This may prevent the formation of multiple small loculated abscesses.

The treatment of obstructions in the cerebrospinal pathway should be by intermittent or continuous drainage of cerebrospinal fluid and open operations should be reserved for the stage of fibrous adhesions and clear fluid. Measurement of the intracranial pressure,

the abundance or scarcity of lumbar spinal fluid, and the spread of an antibiotic introduced intraventricularly or intraspinally will help make the diagnosis of the site of obstruction. It can be determined precisely only by the judicious use of a small amount of air by ventriculography or encephalography.

In tuberculous meningitis there is usually a large deposit of granulation tissue in the basilar cisterns about the peduncles and chiasm. Cairns[1] reported upon the treatment of 93 patients with tuberculous meningitis by streptomycin intrathecally and intramuscularly over long periods of time. The elaborate tissue response in this type of meningitis makes the problem of treatment more complicated and calls for something more than an agent which will destroy bacteria. Their results with tuberculin injected intrathecally under carefully controlled conditions have been encouraging in that tuberculin seems to result in lysis of the exudate and under those circumstances streptomycin is a better remedy than has been supposed.

The important role which surgical methods can play in the diagnosis and treatment of meningitis is considerable, but they are always subsidiary to the main problem which is one of careful bacteriological identification of the offending organism and the specific use of the proper chemotherapeutic agents

[1] Cairns, H., Smith, H. V., Vollum, R. L.. Tuberculous Meningitis, Jour. A.M.A., 144, 92, 1950

CHAPTER V

INFECTIONS AND TUMORS OF THE SKULL

In a certain number of patients changes in the cranial bones give unmistakable evidence of a tumor of the brain and/or its membranes. Infection of the central nervous system may follow infection of the skull bones or vice versa. However, there are other bony changes in the skull which are not concerned with its contents.

Osteomyelitis.—It is probable that secondary infections of the bones of the skull following direct trauma to the scalp and skull have been observed from the earliest times,[1] but the present infrequency of

[1] In 1768 Percival Pott described two types of "bone contusions." First, a localized bony necrosis on which inflammation was engrafted and due to the actual implantation of infected material in the outer table of the skull through an open wound; and second, a local necrosis and suppuration with the overlying scalp intact. This latter condition came to be known as "Pott's puffy tumor."

osteomyelitis of the skull in spite of an increase in the number of scalp and skull injuries, is due to the prompt and careful treatment of potentially infected wounds. By far the largest number of cases which are now observed are the result of an extension of infection from the accessory nasal sinuses, and mastoid, or are hematogenous in origin.

The frontal, parietal, and temporal bones are most commonly involved; the frontal bone as a complication of disease of the frontal and maxillary sinuses and the orbit; the parietal and temporal bones as the result of a spread of infection from the ear. Osteomyelitis of the skull complicating otitis media and mastoid disease does not occur as commonly as it does after nasal accessory sinus disease. This is due to the anatomical characteristics of the temporal bone, in which the diploë between the tables of the skull is less prominent than in the other bones of the skull. In our own group of 37 cases, 6 were traumatic in origin, following as it did after a skull fracture and scalp lacerations which became infected; 5 cases occurred after a frontal and maxillary sinusitis; 2 followed a mastoid infection; another patient, an infant aged seventeen months, developed a frontal osteomyelitis after an infection in the scalp of the forehead due to a lead pencil puncture wound; another patient had a chronic luetic osteomyelitis; 1 patient developed an osteomyelitis of the skull following scarlet fever and meningitis; 1 after a parotid gland abscess. 2 occurred after tantalum insertions, and 6 had de-

veloped an osteomyelitis in a craniotomy wound performed in error when an intracranial abscess was discovered at operation instead of a tumor. In the majority of these patients the osteomyelitic process was well localized, but in 2 cases an extensive spreading osteomyelitis involved practically all of the accessory nasal sinuses.

Regardless of whether osteomyelitis of the skull follows direct trauma or the spread of an infection from sinusitis or mastoiditis, the anatomical characteristics of the bones of the skull play an important rôle in its development.

Between the outer and inner tables of the skull lies a layer of cancellous bone, the diploë. In this layer are numerous venous channels which drain directly into the supraorbital veins and the lateral and sphenoparietal sinuses. The arterial supply to the bones of the skull comes from the dural vessels and the small arterioles of the pericranium. Since the time of Percival Pott it has been thought that separation of the dura mater, or the pericranium, from the tables of the skull predisposes to the formation of a local necrosis of bone by the consequent loss of its blood supply. In addition to this factor, it has been shown in many instances that a thrombophlebitis of the diploic veins occurs in advance of the disease of the bone. The infection travels, therefore, against the flow of blood in the veins and extension is materially aided by the venous stasis which is present.

The course of osteomyelitis is so similar regardless of the method of infection, it appears to us to be difficult to differentiate pathologically between traumatic and non-traumatic types. Clinically, at least, an osteomyelitis may be localized or spreading. To attempt further differentiation according to the etiological factors involved only adds confusion to consideration of the subject.

Localized osteomyelitis is most frequently found following an initial trauma to the skull which produces a local area of lowered bone resistance. The infecting organism may or may not be carried directly into the bone by the injury; in the latter case, the organism may be derived from the blood stream or from hair follicles in the overlying scalp. At any rate the virulence of the organism, the local resistance of the bone and the general resistance of the body bring about a balance which determines the size of the osteomyelitic process The bone immediately surrounding the focus begins to sclerose, and there is a moderate amount of new bone proliferation. Thus, the healing process encircles the area of necrotic, infected bone and ultimately a sequestrum may be formed. It is not difficult to see that under varying conditions and in different individuals a localized osteomyelitis may easily become a rapidly spreading process.

Still another type of localized infection of the bone may occur which is midway between a localized osteomyelitis with its boundaries of sclerosed bone and an extensive spreading osteomyelitis In this circumscribed process, numerous small purulent foci of infection

develop which later become confluent and spread in the surrounding diploic tissue. It is thus possible to have a rather extensive involvement of the diploë without invasion of the outer and inner tables of the skull. The healing process of sclerosis is less marked in this type of the disease, and if the inner table sequestrates, the defect may subsequently be lessened by proliferation of the new bone from the surrounding margins of the inner table. (Fig. 102.)

We have encountered two examples of a rapidly spreading virulent osteomyelitis which extended over almost the entire surface of the

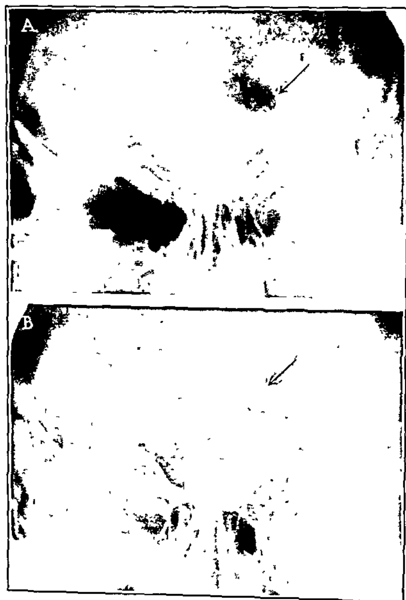


FIG 102.—Osteomyelitis of the skull: (A) roentgen-ray film of the skull which

cranium. This infection followed a pansinusitis with a septicemia and spread through the diploic spaces and venous channels which had become thrombosed as a result of a phlebitis. The extension, as is usually the case, was toward the vault rather than the base of the skull. Before the use of chemotherapy this proved to be an overwhelming infection which was fatal to the patient.[1]

Sequestra vary in their size and shape according to the type of osteomyelitis which exists. In the sclerosing localized type, the sequestrum resembles an elongated island of bone which may remain attached at one end. Circumscribed osteomyelitis is more likely to produce sequestra of the fragmented inner table. Spreading osteomyelitis, when it does not prove fatal, produces a sequestrum which is outlined by the spread of infection along the diploic veins. The sequestrum then becomes invaded with more marked destruction of the outer than the inner table, probably because of the better vascular supply of the latter from the vessels of the dura to which the sequestrum remains attached except at its periphery.

The symptoms of osteomyelitis of the skull may be purely local or they may be characteristic of the general signs of septicemia plus the more localizing symptoms of intracranial involvement. The earliest diagnostic sign may be only a puffy, edematous swelling between the bone and scalp which may be only slightly tender. In more virulent infections of the bone, headache, tenderness of the skull to pressure, vomiting, apathy, and general toxic symptoms may be present. In general, one may say that the presence of complications, such as an extradural abscess, an intracranial abscess or a meningitis, dominates the clincal symptomatology. The development of osteomyelitis of the skull and its consequent complications are well illustrated by the story of a seventeen months' old infant.

While playing with his sister, he was struck in the skin of the forehead with the sharpened point of a pencil. A definite puncture wound was not visible, and little thought was given to the injury by the parents. Within a week a large subcutaneous abscess was incised and drained in the midfrontal area This wound continued to drain, and the child became more irritable and definitely more toxic. The frontal bone was tender, but nothing more could be made out upon careful physical and neurological examination A roentgenray film showed a typical osteomyelitis of the frontal bone. It seemed quite evident that an extradural abscess might also be present The diseased bone was removed and the underlying dura was rough and covered with fungus-like, suppurating granulation tissue. Quite unexpectedly thick, yellow pus welled up from the underlying brain and an enormous frontal lobe abscess was emptied with the suction apparatus For three weeks the child improved

[1] The reader is referred to the excellent articles by Adelstein, L. J., and Courville, C. B Arch Surg., 26, 539, 1933, and Wilensky, A O Ibid, 27, 83, 1933, for an exhaustive consideration of the pathogenesis and pathology of osteomyelitis of the skull.

remarkably, only to succumb suddenly from an intraventricular hemorrhage.

The onset of osteomyelitis of the skull may be so insidious that suspicion of its development does not arise for an appreciable interval. In the diffuse form of this infection, headache is generalized and hard to localize. It is generally more severe at night, as is true of all pain of bony origin. Discrete, doughy swellings may appear, and the scalp overlying them becomes red and tender. These symptoms are quite characteristic of subperiosteal abscesses.

Extradural abscess, meningitis, intracranial abscess, and thrombosis of the many dural venous sinuses are the most common complicating factors in osteomyelitis of the skull. All of these conditions are difficult to treat, and their serious nature increases the mortality of the disease tremendously.

The treatment of this infection is somewhat controversial, in part due to the difficulty of definition and agreement in discussion of any one clinical entity. Each case is almost a law in itself and it seems illogical to take the extreme position that surgery is contraindicated under all circumstances and that more conservative methods of treatment should always be employed. As is usually the case, a more conservative middle ground is the most logical position to assume.

Certainly everyone can be in agreement that a great deal has been done and can be done to prevent infections of the skull. As has been stated, the immediate and proper care of all scalp wounds is most important. It is hoped that the day of applying a brilliantly colored antiseptic solution to the wound and the introduction of large silkworm-gut sutures through matted, bloody hair has passed. Equally as important, however, is adequate treatment of accessory sinus disease under surgical conditions which should be as meticulous in sterility as any other operative procedure. The introduction of probes, which pierce diseased bony walls with more ease than may be realized, should be looked upon as dangerous and in many instances unnecessary. In addition to these prophylactic surgical measures, the patient should be treated systemically with penicillin and/or the sulfonamide drugs. Blood transfusions are very effective in maintaining the patient's general resistance.

In discussing active surgical treatment, one can only indicate certain lines of procedure which may guide the choice of methods. Those cases with the clinical symptoms of an acute, virulent general infection lend themselves poorly to treatment, and in our own experience this is true also of the spreading type of osteomyelitis. In the majority of our patients, the process has been localized, and we have removed the necrotic and infected bone widely. We have been fortunate in having had no fatalities following operation with

the exception of one case with an acute spreading osteomyelitis, who also had an extensive extradural abscess, and the patient with a large frontal lobe abscess referred to above. However, in the series of cases reported in the literature the mortality rate is very high.

It has been shown that in patients who recover from osteomyelitis of the skull, regeneration of the involved bone takes place. In the stage of repair, there is a rapid multiplication of the connective tissue elements at the site of the infection. After a short time some of the connective tissue cells become differentiated and assume the characteristics and functions of mature osteoblasts. There results an interlacing network of uncalcified osteoid tissue; and after calcium permeates this tissue, bone trabeculæ become evident. It has been known for some time that there is a lack of bone regeneration in the burr hole openings and between the edges of the usual osteoplastic craniotomy unless bone dust or particles are left behind. In some manner, infection plays a rôle in the stimulation of the factors responsible for bone regeneration.

As is true elsewhere in the body, the roentgen evidences of osteomyelitis of the skull are late in making an appearance. A week to ten days following bone infection, a small area of rarefaction which may be impossible to differentiate from a venous lake may be seen. In other cases, there may be a small area of only slight demineralization which blends with the surrounding bone. Later the small area of rarefaction increases considerably in size or the demineralized area appears moth-eaten Various sized, irregular and dense sequestra become evident later and stud the affected area which gradually merges with normal bone. Healing occurs by the regeneration of bone and the area becomes irregularly dense If the infected bone has been removed surgically, then a cranial defect with smooth, rounded edges resembling normal bone will be seen.

Spreading osteomyelitis presents a different roentgen-ray appearance. If the infection spreads by continuity, the area involved appears to be studded by minute foci of rarefaction; and as it progresses, these enlarge and coalesce to produce the characteristic moth-eaten appearance. Deprived of a blood supply, small irregular and dense sequestra appear, but in the advance the zone of rarefaction of bone will be found. If the infection spreads along the diploic channels, the dissemination is more rapid so that early, small areas of rarefaction of bone appear along the course of the diploic channels These areas resemble venous lakes but are usually smaller and follow the vascular channels more closely These smaller areas of involvement serve as foci from which larger areas are affected. When spread occurs along the diploic channels, the tendency for osteomyelitis to extend upward toward the vertex of the skull is well illustrated. An

area of osteomyelitis may appear at some distance in the skull from the primary focus as the result of a thrombophlebitis, but the roentgen-ray characteristics are similar. It is difficult to determine, in a well-advanced case, whether the irregularly shaped particles of viable bone exist between areas of rarefaction or whether they are sequestra. (Fig. 103.)

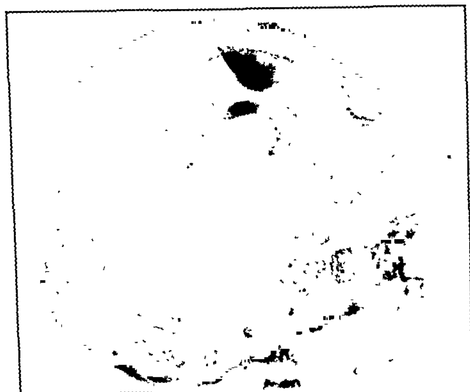


FIG 103.—Osteomyelitis of the skull. Roentgen-ray film of skull which shows a spreading osteomyelitis with areas of sclerosis and sequestra. Partial removal of the infected bone has been effected by surgery.

Tuberculosis of the Skull.—Osteomyelitis, cephalhematomas, and even tumors may be simulated very closely by tuberculosis of the flat bones of the vault of the skull.[1]

Usually attention is first drawn to the head by the appearance of a swelling which is at first only slightly elevated, round, firm, and appears fixed to the bone. This subperiosteal abscess enlarges gradually, but the skin and other soft parts remain freely movable over it, and the skin is quite normal in appearance. Later, as the subperiosteal abscess perforates the periosteum, a cold abscess is formed in the soft tissues; and although it is quite soft in the center,

[1] An excellent description of this condition, which has undoubtedly been overlooked many times, has been written by D. C Strauss in Surg., Gynec and Obst., 57, 384, 1933

the margins are firmly attached to the bone. If the skin over the abscess becomes adherent, inflamed and finally ruptures, a typical tuberculous sinus from which pus continues to discharge is formed.

Tuberculosis of the bones of the skull occurs most frequently in children or in younger people before the age of twenty years. The disease is most commonly secondary to a tuberculous infection elsewhere in the body, and the infection reaches the skull through the blood stream. Tuberculous cervical lymphadenitis, lung, or bone and joint tuberculosis are the most frequent sites of the primary pathology. The disease begins in the cancellous tissue of the diploë; and although two types of pathology have been described, it is probable that the changes are circumscribed in the early stages and later become quite diffuse. The frontal and parietal bones of the skull are more commonly affected because of the presence of a larger amount of cancellous tissue.

Cephalhematoma.—Extravasation of blood in the various layers of the scalp and skull will present different clinical and pathological aspects dependent on the location of the actual bleeding. If the effused blood is not absorbed or does not become organized, the surrounding connective tissue as a rule tends to encapsulate it, thus creating a cyst formation. Due to special anatomical conditions, *i. e.*, the peculiar structure and the circulatory system of bone, which allow only a slight possibility for compensatory adaptation to circulatory disturbances, bleeding into the spongiosa or corticalis of the bones of the skull is apt to produce cyst formations which result in unusual pathological and clinical pictures.

Four such cases were reported by us in 1934. In 2 patients, the cyst formation was found between the skull and the pericranium; the other 2 patients presented a cyst cavity in the diploë of the frontal and parietal bones. Since then we have observed other patients with the same lesion.

Cephalematoma, or tumor cranii sanguineus of the elder authors, consists of a subpericranial blood effusion following the rupture of some of the blood-vessels which run between the pericranium and the skull, and is usually due to an injury during labor Unless infected, ruptured, or associated with intracranial hemorrhage, a cephalhematoma does not produce any clinical symptoms except for the tumefaction. Effusions of the blood beneath the pericranium are limited and outlined by the lines of sutures because of the marked adherence of the pericranium to the bone along the suture lines (Fig 104.)

Depending on their size, cephalhematomata are usually absorbed in from two weeks to three months. The entire process of resorption may, however, be completed in six weeks. In rare cases, a cystic

condition may result, the hematoma remaining fluid in its center, clotting and organization having taken place only about the periphery; and in this state a swelling may persist throughout life. When absorption of the subpericranial blood effusion is delayed longer, the swelling may, according to Virchow,[1] become surrounded by a ridge of new formed bone deposited along the elevated pericranial edge. At the same time the surface of the denuded bone also becomes the site of new bone formation. The blood effusion is slowly resorbed; and when this finally occurs, only a flat, uneven, exostosis marks the previous location of the cephalhematoma. This may sometimes lead to a permanent extracranial deformity.

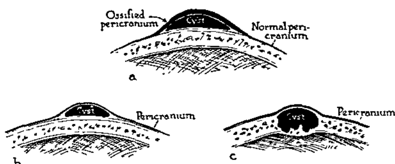


FIG. 104.—Location of cystic formation in cases of: (*a*) ossified cephalhematoma; (*b*) subpericranial blood effusion; (*c*) osteitis fibrosa of the frontal and parietal bones.

Chorobski and Davis[2] described such a case in which the complete absence of a history of an obvious trauma during the delivery or of any systemic causes which could explain the occurrence of the cephalhematoma was noteworthy. However, the lesion was situated in the occipital region which was the leading portion of the head at delivery, and consequently a traumatic origin of the cephalematoma in this case was most likely.[3]

From specimens obtained piecemeal at the time of operation, it is difficult to reconstruct beyond doubt the actual pathogenesis of a given lesion. It may be, however, reasonably assumed that the sequence of events in our case was the following: Due to an injury

[1] Virchow, R: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1, 130, 1863.

[2] Chorobski, J., and Davis, L: Cyst Formations of the Skull, Surg., Gynec. and Obst, 58, 12, 1934.

[3] Here belong, most likely, some of the cases reported in the literature under the name of "sinus pericranii" (Stromeyer). Under this and other terms have been reported traumatic collections of fluid blood under the pericranium, the extracranial sac communicating with one of the dural sinuses either directly or through a venous channel. The pathology of this condition is not unique, however, the "sinus pericranii" representing therefore rather a symptom complex than a pathological entity.

sustained during the delivery, or after, there was a rupture of blood-vessels running between the pericranium and the skull with an effusion of blood between the external table of the skull and the pericranium. Encapsulation and partial organization of the blood effusion followed. It could not be demonstrated where the process of ossification of the outer layers of the hematoma actually started, but the presence of blue colored inclusions of osseous tissue—which is an accepted sign of its primitiveness—and of adult, red stained bony lamellæ, situated more peripherally from the hematoma, pointed toward the pericranium as the origin of the process.

The clinical and pathological picture of another of these cases was somewhat obscure. There was a definite history of trauma which preceded the accidental disclosure of a "lump" in the right fronto-parietal region. The recognition of this lesion was followed a few days afterward by a massive swelling of the scalp on both sides which reached even the right cheek. The appearance of the massive swelling which receded in one week was unaccompanied by a rise of temperature or any other serious systemic reaction except for a dull headache. Previous to admission of the patient to the hospital, the "lump" was incised and it was found at that time to contain a small amount of watery fluid. At the operation a cyst was encountered, located between the skull and pericranium, containing masses of dark clotted blood. Microscopic examination of the cyst wall showed the presence of fibrous connective tissue with a considerable amount of hemosiderin deposits scattered throughout the whole of the stroma. There was also an infiltration of the tissue by round cells and plasma cells, polymorphonuclear leucocytes being absent. The external table of the skull underlying the cyst was found excavated. (Fig. 105.)

It could be assumed that the pathological condition in this case was caused by repeated small traumas to the head which produced a blood effusion between the skull and pericranium.

This extravasation became encapsulated in a connective tissue wall, as evidenced by the presence in it of large quantities of blood pigment deposits Following the disclosure of the lesion by the patient a mild inflammation of some of the loose layers of the scalp occurred —most likely of the subaponeurotic space—which, subsiding clinically, left behind it its histological manifestation in the form of the above mentioned round-cell and plasma-cell infiltration. The presence of hemosiderin deposits in the cyst wall, showing no abnormal vascularity and no retrograde changes in its stroma, speaks against the assumption that the cyst formation and the blood pigment deposits were the results of degenerative and hemorrhagic changes in an already pre-existing pathological lesion. (Fig. 106.)



FIG 105.—(A)Roentgen-ray film of the skull in a patient ... trauma. Note the circular de ... suture line (December 18, 192 ... patient, which shows progressive diminution in size of bony defect following removal of cyst (May 20, 1933).

The dark clotted blood masses found in the cyst cavity at the time of operation were most likely the result of the trauma of the incision into the cyst. Only a serum-like fluid was obtained at that time. Indeed, if the clotted blood came from the original trauma, one would be at a loss to explain why the hemorrhage was not organized instead of being encapsulated. The presence of the serous fluid in the cyst cavity, on the other hand, is in accord with many observations that organization of a hematoma does not take place when connective tissue comes in contact with blood serum.

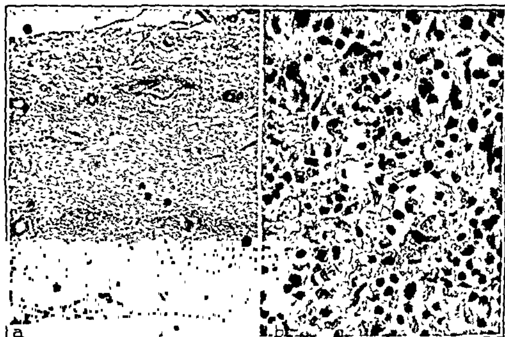


FIG. 106.—(*a*) General structure of the cyst wall showing perivascular infiltration; (*b*) hemosiderin deposits in an area of lymphocytic infiltration.

The depression in the external table of the skull underlying the cyst formation was due probably to a pressure necrosis. For this possibility and against the assumption of a primary bone lesion was its smooth surface, the absence of any pathological manifestation at its borders, distinct filling in and regeneration of bone after the cyst had been removed, and the present excellent condition of the patient as well as the negative roentgen-ray examination of her skull.

Osteitis Fibrosa Cystica.—Osteitis fibrosa cystica[1] is not commonly found in the skull. Geschickter and Copeland reviewing the records of the surgical laboratory of the Johns Hopkins Hospital extending over a period of thirty-five years found that in the purely

[1] Excluding osteitis fibrosa of the upper and lower jaw, temporal fossa, and the epulis of the alveolar border.

membranous portions of the calvarium (the frontal and parietal bones) not a single instance of these lesions has been recorded.

In the one patient we have seen with this condition, the course of the disease was that of chronic, symptomless lesion of the skull. One area of swelling was just anterior to the frontoparietal suture and the other just posterior to it. The first "lump" had been noticed on his forehead in 1919 and had grown slowly and steadily for eight years. It was not until six years after his first operation that he noted a second bony protuberance in the midparietal region. Roentgenologically, the lesions presented some similarity to pictures observed in cases of hemangioma of bone. In the latter condition, however, the trabeculations visible on the roentgen-ray films arise in a common center and radiate out from the plane of the bone. The loculations in hemangioma, in comparison with giant-cell tumor or bone cyst, are somewhat smaller and the cortex of the bone is not expanded as in bone cyst but is partially eroded. (Fig. 107.)

Histologically, the examination of the tissues removed at the operations revealed pictures characteristic of an osteitis fibrosa. A transformation of the bone marrow into fibrous tissue, characterizing osteitis fibrosa, may be followed by regressive changes leading to bone-cyst formation or by progressive changes giving rise to a so-called giant-cell tumor. Cystic softening of the latter and spontaneous disappearance have been observed several times so that it is not always easy to know exactly how the given bone cyst developed. (Fig. 108.)

Whereas the histological examination of the specimens obtained at the first operation upon the patient showed, generally speaking, a picture of a granulation tissue with new-bone formation and evidence of bone destruction—a picture typical of bone cysts occurring in the course of an osteitis fibrosa—the microscopic structure of the tissues removed at the second operation was quite different. The most striking feature at a second operation, at which time a second tumor mass was removed, was the presence of large groups of oval, round, or polyhedral cells with large centrally located nuclei, containing one or two distinct nucleoli, slightly granular cytoplasm with distinct cell borders and drawn out filamentous cytoplasmic processes connecting many of the cells. Most of the cells contained vacuoli, but due to the paucity of material the presence or absence of mitochondria could not be ascertained. Clusters and sheets of these cells were separated by a distinctly fibrillary stroma, stained red with Van Gieson's method. The general morphology and arrangement led us to consider these cells as young osteoblasts, ready to build up new bone. The presence in the cellular areas as well as in the dense fibrous tissue portions of the cyst walls, removed at both operations,

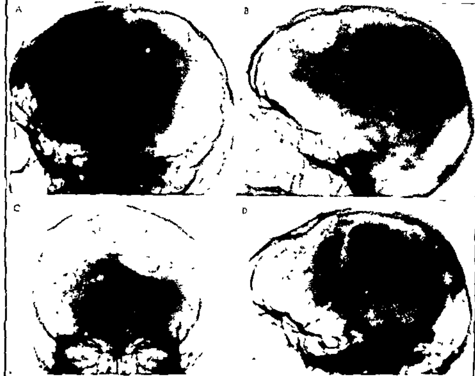


FIG. 107.—Osteitis fibrosa cystica of skull: (*A*) Note the separation of the external and internal tables of the skull. (*B*) Recurrence of cyst of skull after removal of process shown in (*A*). Note the honeycombed appearance in the bone. (*C*) Antero-posterior film which shows multiple cysts in the area affected (*D*) Roentgen-ray film following third operation for osteitis fibrosa cystica. A second area had developed after removal of the primary area of involvement.



FIG. 108 —Osteitis fibrosa cystica (*a*) To demonstrate the general structure of [illegible] cells resembling [illegible] of [illegible] but are connected with each other by protoplasmic expansions.

of multiple elements of the hematopoietic series, is not surprising in a lesion going on in the bone marrow or in close relation to it. (Fig. 109.)

A granular, or "salt and pepper," appearance of the skull is said to be characteristic of osteitis fibrosa, but this is a questionable roentgen-ray diagnostic sign because the same appearance may be seen upon normal skulls which contain many diploë. It is important, therefore, to find cystic defects with clear cut borders before the diagnosis can be made. The "fish" type of vertebræ, with concave superior and inferior surfaces, produced by decalcification and pressure of the intervertebral disc on the body of the vertebra; demineralization of all of the bones so that they do not appear as dense as

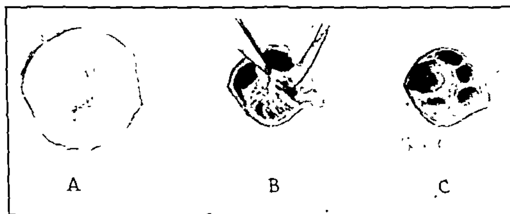


Fig. 109.—Sketch made at the time of operation in a case of osteitis fibrosa cystica showing: (*A*) Bony dome covering the cyst cavity; (*B*) dissection of the capsule; (*C*) multilocular base of the cyst cavity.

normal on the roentgen-ray films; a high blood calcium and low blood phosphorous content; the frequent association of renal calculi; thinning of the cortex of the long bones and prominence of the trabecular markings; multiple cysts and frequent pathological fractures are other roentgen-ray diagnostic aids.

Myeloma.—The true myeloma, *i. e.*, a neoplasm arising from the elements of hematopoietic series, is a rare condition with a rapid course. It affects patients between the ages of forty and sixty years and is characterized by the multiple areas of destruction of cortical bone, protrusion beneath the periosteum and finally infiltration of the surrounding structures. Pains in the affected bone, cachexia and emaciation, secondary anemia, enlargement of the spleen, hyperplasia of the regional lymph nodes, presence of the Bence-Jones reaction in urine, alterations in the blood picture of the patient and metastases to internal organs are usually present in cases of myeloma. (Fig. 110.)

The roentgen-ray films show punched out areas in the skull, the edges of which are discrete. These defects are multiple, and the areas of destruction vary in size and shape. There is no evidence of *any effort at new bone formation. Other bones, particularly* those of the pelvis, also show the same well-defined bony defects. In a recent patient the first indication of difficulty was the development of a large tumor mass in the spinous process of the fifth cervical vertebra. This proved to be a myeloma and other evidences of the multiplicity of this tumor were found in the skull and pelvis.

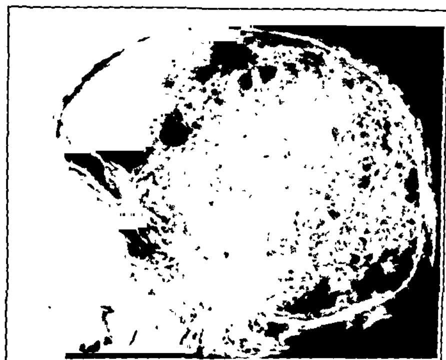


FIG 110 —Multiple myeloma of the skull Roentgen-ray film of the skull which shows multiple areas of destruction which are "punched out" and whose edges are discrete. There is no evidence of any new bone formation.

Meningioma.—A localized thickening of the skull should immediately raise the suspicion of an underlying intracranial meningioma Attention has already been called to the frequency with which hyperostosis of the bone occurs over these fibroblastic tumors of the meninges (Chapter III). As a meningioma grows, its cells penetrate the dura mater and invade the overlying bone throughout which they spread. The skull may be eroded in areas, but in the main the tumor exerts a stimulating influence and an hyperostosis is formed. The new bone originates from the skull and not from the tumor and furnishes, therefore, an example of osteoplastic invasion of bone by a mesoblastic tumor which originates outside the skeleton. Invasions

of bone by other non-bony tumors of mesoblastic origin, whether they occur by direct extension or by spread through the blood stream, are destructive in nature and no new bone is formed.

Osteoma.—We have observed 14 primary osteomas of the skull, and in each instance the history of the growth of the tumor was very slow and was definitely associated with a specific, single trauma to the skull at the site of the neoplasm. The distinction between an inflammatory reaction in the course of which bone is formed and a true bony tumor is often very difficult to make, but in general it may be said that in the latter case new bone is the essential and not an accidental product. In one instance an osteoma occurred at the location of a fracture of the occipital bone produced by a direct blow twenty-four years previously, and at operation the origin of the bony growth could be verified in the irregularity still present in the outer table. In another patient the tumor had grown slowly for a period of ten years, following a blow to the temporal bone by a tennis racquet. It may be assumed, although not proven, that trauma stimulated the new production of bone. (Fig. 111.)

The symptoms in each of these patients were those of discomfort due to the size and prominence of the bony protuberance; occasional tenderness of the scalp over the tumor; and headaches which the patient attributed, no doubt erroneously, to the growth. In no case were there symptoms of involvement of the nervous system.

In 2 of these cases, the growth was a hard, firm, eburnated, spherical mass, the boundaries of which were somewhat difficult to determine since no cancellous diploic layer was present. In a third case, the osteoma was similar in shape and type, but was attached by a very narrow bony pedicle to the outer table, a very unusual occurrence. The tumor itself contained no cancellous bone, or blood-vessels, and was covered by the periosteum to which it was not attached. In no instance was there any evidence whatever of an osteoblastic process, but their unsightliness and the nervous irritability which they produced in each of these patients provided sufficient indications for their complete removal.[1]

In contrast to these benign bony tumors, the roentgenographic appearance of which is decidedly circumscribed, there are less well classified tumors which are apparently primary in the skull and are equally benign, but which immediately suggest the type of hyperostoses which occur over a meningioma. Unfortunately, these tumors

[1] Although other types of osteomas occur which have been termed spongiosa and medullare because they resemble the structure of the diploë or contain large lacunæ, they are much less frequent than the eburnated type such as are here reported. Orbito-ethmoidal osteomas have been referred to in Chapter III and have been well described by Cushing

in some areas and eroded and very vascular in others. The tumor did not penetrate the thickened dura mater.

The roentgenographic appearance of the skull in Paget's disease is quite characteristic. The marked, asymmetric deformity of the cranium and the tremendous increase in thickness are striking. There is evidence of both osteosclerosis and osteoporosis which produces the typical "nigger wool" appearance of the skull. In the long bones, thickening of the cortex is evident and the trabecular markings are very prominent.

Lipoma.—Often tumors of the skull are very difficult to diagnose from their gross or roentgen-ray appearance. An example of this difficulty in diagnosis is furnished by a woman, not at all ill, who complained of the discomfort which accompanied an enlargement of the left frontal bone.

Fifteen years before, the mass had been operated upon elsewhere and an incomplete removal had been performed. The tumor was firm except in its center where a doughy-like consistency was palpable. The roentgenographic appearance was one of an increased production of new bone plus an extensive area of erosion. Although no neurological symptoms were present, a preoperative diagnosis of meningioma with bony changes in the skull was made. Upon operation, a rather large, yellow fatty tumor was found which had eroded the bone and was enmeshed within it. This erosion was produced by pressure rather than by invasion of the tumor. At the periphery of the tumor *the skull was enormously thickened as the result of reactive stimulation by the* tumor, and the diploic layer had entirely disappeared. The entire mass was completely removed and on microscopic section we were greatly surprised to find a typical lipoma The tumor had not penetrated the inner table, and the dura mater was found intact.

Just where such a lipoma might originate in the flat bones of the skull is difficult to say, unless from the diploë. Sarcomatous degeneration of lipomas originating in the medullary cavity of long bones is not uncommonly seen. Of course, lipomas within the cranial cavity are well known in situations in which small collections of fat are normally present, such as the surface of the corpus callosum, the base of the cerebrum, the brain stem, and the roots of the cranial nerves. Areas of bone formation in lipomas are quite common, but this case is of particular value in emphasizing how unexpected and bizarre apparently simple tumor masses may prove to be.

Hyperostosis Frontalis Interna.—This is a benign hyperostosis of the inner table which occurs in about 5 percent of all skulls but predominantly in women. The surface of the hyperostosis presents a scalloped appearance and between the prominences deep vascular furrows are visible. It has been suggested that endocrine and metabolic disturbances are correlated with this finding but the occur-

rence of various symptoms in our experiences cannot be related to this bony change.

Dyscrasic Hyperostosis.—In *rickets*, the cranial bones are thin and osteoporotic but after treatment they become abnormally thick; excess bone is laid down irregularly and usually the frontal and parietal protuberances are prominent. A fine granular, or salt and pepper, appearance of the skull due to osteoporosis characterizes *renal rickets*. In *erythroblastic anemia* the cranial bones are thicker yet more porous than normal. The thickening is due to proliferation of the diploic layer at the expense of the outer table which may become so thin as to be unrecognizable. The radiolucent diploic bone and the disappearance of the outer table produces osteoporosis with a spongy appearance. Perpendicular striations in the enlarged diploic zone extend from the inner table to the periphery of the skull and simulate cropped hair standing straight up on the scalp. In *sickle cell anemia* the changes are similar to those just described but the hyperostosis is usually greater in the parietal bones than elsewhere.

Inflammatory Hyperostosis.—In the early stages of acquired *syphilis* there is a localized thickening of the periosteum and slight absorption of the outer table in the region of the frontal protuberance. Later, circumscribed or diffuse areas of rarefaction with a motheaten appearance of the outer table are seen and even larger pieces of bone may be isolated from their surrounding area. In tertiary syphilis, single or multiple, sharply outlined, kidney-shaped defects develop with or without thickening of the surrounding area. In congenital syphilis similar changes in the skull are seen. *Xanthomatosis* of the Schüller-Christian type represents a form of lipoidosis which usually starts in the skeleton and especially in the skull. The disease usually occurs in childhood and although the pathogenesis is unknown, it is generally accepted that there is a faulty metabolism resulting in the storage of cholesterine-containing lipoids in the reticulo-endothelium of the bones and other organs.

Metastatic Neoplasms.—All of these lesions must be differentiated from the metastatic malignant tumors, particularly carcinoma. The primary tumor is most commonly found in the breast, thyroid gland or prostate gland. The roentgenographic appearance of these metastatic lesions is such that they may be confused with multiple myeloma. The bony defects in the skull are irregular in size and shape and are produced by erosion which leaves an irregular rough edge quite contrary to the smooth, discrete edge found in myeloma. Sometimes there is evidence of bony sclerosis in the metastatic lesions of carcinoma so that the lesion shows evidence of both destruction and production. (Fig. 113.)

We have had an experience with a malignant tumor of the thyroid which produced multiple metastatic lesions of the skull. The largest mass in the parieto-occipital area was about the size of an hen's egg, soft and fluctuant. The bone immediately surrounding this area was raised and sloped upward to the base of the mass. Within four weeks,



FIG 113.—Metastatic carcinoma. Roentgen-ray film of the skull which shows metastases of carcinoma of the breast to the skull. The bony defects in the skull are irregular in size and shape. They are produced by erosion which leaves an irregular rough edge in contrast to the smooth discrete edge found in multiple myeloma

a metastatic growth developed in the left humerus, and two more punched-out areas of decreased density were found in the skull. Given time, these malignant tumors of the skull invade the cranial cavity and progressive neurological symptoms are produced which eventually lead to death (Fig. 114.)

Involvement of the skull in *Hodgkin's disease*, *lymphosarcoma* and *leukemia* is also osteolytic and resembles the lesions just described.

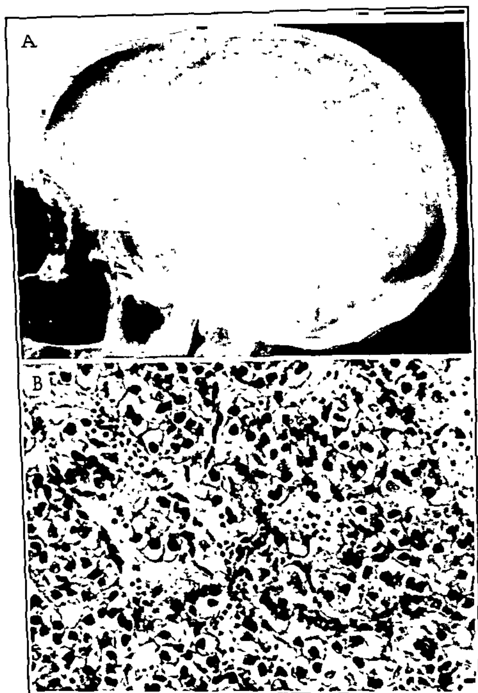


FIG. 114.—Metastatic hypernephroma: (*A*) Roentgen-ray film of skull which shows metastases of a hypernephroma to the skull. Note the irregular strands of bone interlaced in the area of erosion. (*B*) Photomicrograph of hypernephroma which shows the clear cytoplasm of the cells. (× 410.)

CHAPTER VI

SURGICAL LESIONS OF THE CRANIAL NERVES

TRIGEMINAL NEURALGIA

"In cases of singular difficulty and obstinacy, it is natural for us to be inquisitive into their causes and their nature; unsuccessful experiments sometimes lead the way to instruction; and we ought never to cease investigating the most abstruse recesses of Nature, nor at the same time forget the narrow limits of our capacity, and the danger of presumption."—JOHN FOTHERGILL.

THE functions of the cranial nerves are impaired in many diseases of the central nervous system but several of them are subject to pathological conditions, the symptoms of which are now recognized as clinical entities. Perhaps the most striking example of these is *trigeminal neuralgia*,[1] a disease of unknown etiology, characterized by paroxysmal excruciating pain of sudden onset in the area of distribution of the fifth cranial nerve, which may be initiated by the stimulation of dolorogenic zones Until it was recognized as a clinical entity, trigeminal neuralgia was a part of that large group of cases diagnosed rather indefinitely as facial neuralgia.

This disease occurs most commonly in patients in the fifth, sixth, and seventh decades of life. The youngest patient in a series of 556 cases was aged twenty-one years and the oldest eighty-three years. Of these 556 patients, 281 were females and 275 were males

Since the thirteenth century it has been thought that carious teeth played a rôle in the etiology of trigeminal neuralgia. Upon the two pillars which face the south aisle of the choir within the Wells Cathedral in Somerset, there are three carved figures which are illustrations of persons suffering with violent facial pain. One is of a monk with an open mouth, with tongue protruding and his distorted features indicating severe agony and the others are of individuals obviously suffering with toothache. These figures bear witness to the offerings to the Cathedral funds by pilgrims who came to the tomb of Bishop Button. The contributions which helped furnish the Cathedral were made at his tomb by sufferers from toothache and neuralgia who came in large numbers to be cured by his saintly influence. The Bishop's reputation for curing toothache and neuralgia may be explained by the fact that although he died in very old age, he had a perfect set of thirty-two teeth with no trace of caries or

[1] Also known as *tic douloureux, trifacial neuralgia, Fothergill's disease, epileptiform neuralgia, neuralgia major and prosopalgia.*

discoloration. Since the Bishop's day, the treatment of trigeminal neuralgia is usually shared between the dentist and the physician. The pain may appear to originate in a tooth, and though it be perfectly sound, the patient insists upon extraction. Many of these patients have all of their teeth removed in an unsuccessful effort to relieve the pain though no definite etiological relationship can be established between carious teeth or any trauma which might attend their extraction and the pain of trigeminal neuralgia.

Other possible foci of infection such as the tonsils, ears, and sinuses have been thought of as possible etiological agents, but the removal of these infections has never been followed by lasting relief from the pain. The increased susceptibility of some individuals to herpes labialis has also been investigated in relation to the occurrence of trigeminal neuralgia but without definite results.

That many of these patients have advanced signs of arteriosclerosis and that many suffer from hypertension is apparent from a study of any large series of patients, but these instances are balanced by those patients whose blood-pressure is within normal limits and whose arterial system is quite elastic.

In our own cases there have been 9 patients with definite symptoms of multiple sclerosis who had typical attacks of trigeminal neuralgia. Parker[1] has described 4 such cases and in 1 found 2 sclerotic plaques in the pons at the point of emergence of the sensory root on the side affected. The sclerotic process extended outward a short distance within the substance of the root but the Gasserian ganglion and peripheral divisions showed no pathology.

Caries of the petrous portion of the temporal bone, tumors, or aneurysms in the cerebellopontile angle, and osteomas of the middle fossa of the skull, among many other diseases whose number is legion, have been cited in the literature as probable etiological factors but as yet proof is lacking.

That there may be some connection between migrain and trigeminal neuralgia has been indicated by Patrick.[2] In a study of 342 cases of trigeminal neuralgia, Paskind[3] found a history of migrain in the family in 37.7 per cent; in a parent in 30.1 per cent and in the patient in 23.3 per cent of the patients.

Fothergill's original observation and description of 14 cases led him to suggest that the disease was due to carcinoma. Perhaps he

[1] Parker, H. L.: Trigeminal Neuralgic Pain Associated With Multiple Sclerosis, Brain, 51, 46, 1928.

[2] Patrick, Hugh T.: The Symptomatology of Trifacial Neuralgia, Jour. Am. Med. Assn., 62, 1519, 1914.

[3] Paskind, H. A.: Relationship of Migrain, Epilepsy and Some Other Neuropsychiatric Disorders, Arch. Neurol. and Psychiat., 32, 45, 1934.

would view our surmises as to the etiological factors involved with as much dissent.

Microscopic examinations of the Gasserian ganglion, portions of the sensory root and peripheral branches of the trigeminal nerve have been made many times and by many different investigators without finding pathological changes of significance. The lesion in *trigeminal neuralgia*, whatever its nature may be, is not a destructive one because of the absence of degenerative findings in the cells of the ganglion or in the peripheral or sensory root fibers, and the total lack of loss of sensation to any modality in the cutaneous distribution of the nerve.

The vasomotor symptoms which are characteristically present during the attacks of terrific pain have led some to implicate the sympathetic nervous system, but again no definite gross or microscopic pathological lesions have been described.

Tumors or aneurysmal vessels which produce pressure upon the *Gasserian ganglion or the sensory root and cause severe pain in the* face are also accompanied by a loss of sensation or extra-ocular nerve palsies. Such a combination of symptoms should immediately suggest that the case was one which should be diagnosed as some condition other than trigeminal neuralgia.[1]

Though this painful and intractable disease of the face may have been encountered, it was not described as an entity until John Fothergill, a London physician, wrote his monograph, "Of a Painful Affection of the Face" in 1776. So it has become known, among its many other names as Fothergill's disease. Like many other diseases in medicine and surgery, Fothergill's original description has been improved upon but little. He said:

> "This affection seems to be peculiar to persons advancing in years and to women more than to men. I never met with it in anyone much under forty, *but after this period no age is exempt from it.*
>
> "From imperceptible beginnings, a pain attacks some part or other of the face, or the side of the head, sometimes about the orbit of the eye, sometimes the ossa malarum, sometimes the temporal bones, are the parts complained of. *The pain comes suddenly and is excruciating*, it lasts but a short time, perhaps a quarter or half a minute, and then goes off; it returns at irregular intervals, sometimes in half an hour, sometimes there are two or three repetitions in a few minutes.
>
> "The kind of pain is described differently by different persons, as may be reasonably expected; but one sees enough to excite one's compassion if present during the paroxysm.
>
> "Eating will bring it on some persons. Talking, or the least motion of the muscles of the face affects others; the gentlest touch of a hand or a handkerchief will sometimes bring on the pain, whilst a strong pressure on the part has no effect.

[1] Davis, Loyal, and Martin, John. Surgical Lesions of the Paratrigeminal Area. Jour. Am. Med. Assn., 113, 1952, 1939.

"It differs from the toothache essentially in many respects. It affects some who, from age, have few or no teeth remaining."

The onset of the pain of this disease is often sudden and unforgettable. The patient experiences a lightning-like jab of pain in his upper or lower gums, at the side of the nose, in the upper or lower lip, or on the cheek beneath the eye. More rarely the first pain may be felt in the ophthalmic area of supply. When questioned years later patients may describe with vivid accuracy with what tasks they were busied at the time of the onset of the pain. One individual had just sat down at his dining table on Thanksgiving Day; another was in the act of hanging out her freshly washed clothes; and a third startled the audience and players in a theatre by her piercing screams. The initial pains are usually of very short, momentary duration and are described by the patient as "jabs" with a red hot poker or sharp knife. This series of pains may disappear just as suddenly as they came and the patient again feels perfectly well. If the pain occurs in the upper or lower gums, it is attributed rather naturally to a tooth which in the mind of the patient apparently localizes the pain accurately. Days, week, months or years later and though the guiltless tooth may have been removed, the pain just as suddenly returns. The duration of the pain usually increases, it may radiate to another division of the nerve and the paroxysm now has a climax (sometimes likened to the explosion of a bomb within the face) but still the onset and termination are dramatically abrupt.

If the patient is examined carefully, dolorogenic areas, or the trigger zones of Patrick, will be found. These are areas on the skin, on the mucous membrane of the cheek, on the side of the tongue, on the gums, on the upper or lower lip, at the angle of the nose, in the nares or anywhere within the distribution of the fifth nerve, stimulation of which will initiate a typical paroxysm of pain. Not uncommonly the trigger zone may be in the area of supply of one division of the nerve, and the pain may occur in the area innervated by another division. These trigger zones are quite characteristic of trigeminal neuralgia and constitute one of its most prominent symptoms. A light breath of air upon the cheek; the slightest touch on the lip; any movement of the face, such as laughing, talking, eating, sneezing, or blowing the nose, will start the pain. In many cases, patients cannot stand the excruciating pain occasioned by washing or shaving the affected side of the face, and often one may see several weeks' collection of sebaceous secretion and dirt which has been left untouched. It is not uncommon to see patients who are dehydrated and emaciated because of their inability to take foods or fluids without initiating paroxysms of pain, the anticipation of which has seemed unbearable.

Patients behave differently during the attacks. With some, the affected side of the face is screwed up tightly with the eyelids closed; with others the jaw may be fixed wide open and saliva drools from the mouth. In other patients tears stream from the eyes and they perspire freely. Some place a hand firmly on the cheek in an effort to stop the pain; while some place their hand in a protective attitude beside the face but dare not touch it. Others rub the face briskly during the attack and so hard that the skin may be worn off.

Practically every patient is free from paroxysms of pain at night due, no doubt, to the absence of facial movements and quiet. In some, however, the attacks continue night and day and no relief is obtained until the severity of the attack abates and an interval of freedom from the pain returns. These intervals of freedom may vary from complete relief to a condition in which the paroxysms of severe pain have disappeared, and only short, electric shocks of pain with long intervals of freedom between are present. Some patients never lose the sensitiveness of the face even though stimulation of the trigger zones does not bring on the pain, and the attacks are in complete abeyance.

The disease becomes more chronic as the years go by; the intervals of freedom between attacks become shorter although the severity of the pain may have become somewhat less. Invariably the pain persists relentlessly and in former years before modern methods of treatment were known, suicide often terminated the disease. In one instance in this series, the patient, aged seventy-two years, suffered with the pain for thirty-six years before finding complete relief.

The pain of trigeminal neuralgia never radiates across the midline although both of the trigeminal nerves may be affected. Bilateral trigeminal neuralgia is not common, however, and in practically every instance several years elapse before the second side becomes involved. In this series only 3 cases of bilateral trigeminal neuralgia have been observed.[1]

The picture of agony presented by a patient suffering from a paroxysm of pain of trigeminal neuralgia is unforgettable and is not reproduced in all of its characteristics by any other disease, a fact which alone corroborates the diagnosis of trigeminal neuralgia. Stimulation of the trigger zone will produce a paroxysm of pain when the patient is in the throes of an attack; and as the patient commonly locates these areas for the examiner, they are not difficult to find The remainder of the physical examination usually discloses nothing with direct bearing upon the diagnosis.

[1] Frazier, C. H Division of Sensory Root on Both Sides; First Experience in Series of 432 Radical Operations for Major Trigeminal Neuralgia, Jour Am. Med Assn., 87, 1730, 1926, encountered the disease bilaterally 7 times in 1220 cases.

The patient's story of the onset of the pain, the exact location, radiation and character of the pain is so striking and so definite as to be impressive. It is seldom indeed that they do not remember the exact situation and conditions under which the very first attack occurred, and this is an important diagnostic fact.

The pain of sinus disease is not paroxysmal in character; it is a heavy, severe neuralgic type of pain which occurs usually in the later hours of the day, and as the sinus fills with secretion the pain becomes worse. There are no striking intervals of freedom from pain. As Fothergill said, it differs from the toothache in many particulars, but here again the paroxysmal, climactic and explosive character of the pain of trigeminal neuralgia serves to make differentiation rather simple.

Glossopharyngeal neuralgia is a disease similar to trigeminal neuralgia in every respect except that it involves the ninth (glossopharyngeal) cranial nerve. The attacks of pain are alike, as is the onset and course of the disease. The trigger zone is most commonly situated on the anterior pillar of the tonsillar fauces, and stimulation of this area with a cotton applicator will initiate the pain. The pain is felt just below the angle of the jaw on the side of the throat and below the ear; it spreads upward toward the ear and toward the cheek. A more detailed account of this disease will be found in the section of this chapter which deals with glossopharyngeal neuralgia.

Tumors of the Gasserian ganglion, or of the cerebellopontile angle, and aneurysms of the internal carotid artery may produce severe pain in the face; but the pain is of longer duration and does not have such an abrupt onset and termination. Moreover, rather soon these lesions become destructive in nature, and areas of sensory loss to one or more modalities of sensation may be found upon examination of the trigeminal cutaneous distribution. As these tumors progress other cranial nerves become involved or other symptoms of an intracranial tumor appear.

Deep, boring pain in the maxilla and cheek has been ascribed by many rhinologists as due to neuralgia of the sphenopalatine ganglion. Often the pain is of a burning character aggravated by cold wind which strikes the nasal mucous membrane and by eating. In many such patients the pain is localized near the inner canthus of the eye but spreads through the cheek, temple, and side of the head to the back of the neck. Many patients describe their pain as being similar to the radiating sensation experienced when ice cold liquid strikes the roof of the mouth. Cocaine solution may be placed on the mucous membrane of the nose just posterior to the middle turbinate where this ganglion will be affected, and relief from the pain will serve as an accurate method of differentiation.

Though not common, tabetic pains may occur in the distribution of the fifth nerve and differentiation from the type of pain alone may be impossible. We have observed one patient of this kind, but a careful neurological examination of the pupils, reflexes, and sensation pointed to the correct diagnosis. Malignant growths of the face and mouth may, by extension, involve the fifth nerve and its divisions and produce a severe agonizing pain in the face, but differentiation from true trigeminal neuralgia is not difficult.

There is a group of patients who complain of pain in the face, which is not characteristic of trigeminal neuralgia or any of the other common painful affections of the face. Careful search for etiological factors in these cases may not be successful and relief from the pain cannot be secured by any of the common analgesic drugs. It has become the custom to group these cases under the term of "atypical neuralgias of the face" mainly because of the lack of knowledge concerning the correct diagnosis. In all of these patients, in whom the character of the pain may be suggestive of trigeminal neuralgia, the absence of trigger zones makes differentiation possible. Among this group are patients who have severe pain due to a traumatic arthritis of the temporo-mandibular joint. Injection of fibrosing solutions into the joint to reduce its laxity has resulted in relief.

The clinical course of trigeminal neuralgia is characterized by intervals of freedom from the pain which are as abrupt in their onset as is the initiation of the pain. These may vary in length from days and months to years without any definite cause although it is customary for the patient to associate freedom from the pain with the last therapeutic method which has been tried. Invariably, the pain returns to last an indefinite and usually a longer period of time made unbearable by a more agonizing and severe pain. As far as is known, no case of trigeminal neuralgia has ever ceased spontaneously although intervals of many years between attacks have been recorded.

In an occasional case in the early part of its course when the severity of the attacks of pain is slight, medicine may give relief, but in the majority of patients drugs fail to control the excruciating pain of trigeminal neuralgia. The usual analgesics are of little value though in combination with other hypnotics some slight relief may be obtained. Often morphine alone does not help but in combination with hyoscine, temporary relief and sleep may be produced. In some instances, inhalations of trichlor-ethylene may be of help although it has been our experience that as time goes on relief becomes less sure and complete.

Recently there have been reports in the literature tending to show that the administration of vitamin B_1 will permanently relieve the pain of trigeminal neuralgia. Undoubtedly, many of these patients

may suffer from a proven vitamin deficiency and if so, they should be properly treated, but we have a large series of patients who have had to be operated upon because of a recurrence of pain following this type of therapy. Davidoff has given 60 grains (4 grams) of ferrous carbonate twice daily to these patients and states that 50 per cent of the patient's trigeminal pain is relieved. It must be said that many therapeutic methods have been tried and at first thought to be successful often because the patient had a remission coincident with the therapy. In the experience of our patients no medicinal treatment has provided lasting relief.

In 1890, Rose[1] excised the superior maxilla, trephined the base of the skull with the foramen ovale as a center and curetted away the Gasserian ganglion. At about the same time, the older Edmund Andrews, among others, exposed and excised the ganglion through the pterygoid fossa, but as might be expected, these radical operative procedures were followed by an extremely high mortality. In 1891, Horsley[2] suggested and performed division of the trigeminal nerve posterior to the ganglion, and although he stated that "the operation presented no special difficulty beyond that of being very tedious" the patient died seven hours later of shock. In 1892, Hartley[3] and Krause[4] simultaneously advocated an approach through the temporal bone by means of an omega-shaped flap of skin, muscle, periosteum, and bone. This procedure was known as the high temporal operation, in contradistinction to the method proposed first by Cushing[5] and later by Lexer[6] in which the line of incision was somewhat lower. These suggestions concerned only the means of reaching the ganglion to excise of avulse it. Because of the high mortality resulting from avulsion of the ganglion, operative procedures were limited for a time to division of the peripheral branches involved. Such incomplete measures were almost always followed by regeneration of the avulsed nerve and by attacks of pain in one of the remaining branches, so that further surgical measures became necessary.

In 1901, Frazier divided the sensory root posterior to the Gasserian ganglion following a suggestion made by Spiller in 1898 that the

[1] Rose, W.: Removal of the Gasserian Ganglion for Severe Neuralgia, Trans. Med. Soc. London, 14, 35, 1891.

[2] Horsley, V.: Remarks on the Various Surgical Procedures Devised for the Relief of or Cure of Trigeminal Neuralgia (Tic Douloureux), Brit. Med. Jour., ii, 1249, 1891.

[3] Hartley, F. [illegible] cond and Third Divisions of the Fifth Nerve, N [illegible]

[4] Krause, F.: [illegible] des central davon gelegner Trigeminusstamme[illegible] Wchnschr., 19, 341, 1893.

[5] Cushing, H.: Extirpation of the Gasserian Ganglion, Jour. Am. Med. Assn., 34, 1035, 1900.

[6] Lexer, E.: Zur Operation des Ganglion gasseri nach Erfahrungen am 15 Faellen, Arch. f. klin. Chir., 65, 843, 1902.

fibers of the sensory root when divided between the ganglion and their entrance zone into the pons will not regenerate.

Spiller said, "If it could be shown that the sensory root of the Gasserian ganglion does not unite after its fibers are divided, we should have a fact of great importance. Division of this root would probably be a less serious operation than the removal of the entire ganglion and might have the same effect in the relief of pain, but the surgical difficulties might be insurmountable. Experiments on animals to determine whether or not the sensory root of the Gasserian ganglion unites after section of its fibers might result in a lessening of the great mortality now existing in operations on the ganglion."

This is the fundamental principle upon which the present day surgical treatment of trigeminal neuralgia is based.

At about this time, when the mortality of surgical procedures was admittedly high, and when the conclusive evidence of Spiller and Frazier was not yet accepted widely, Pitres and Verger and subsequently Schloesser[1] advocated alcohol injection of the deep foramina of exit of the three divisions of the trigeminal nerve from the skull. The effect of the alcohol is to produce a local destruction, by coagulation, of the nerve fibers at the point of injection. This is followed by degeneration below that point to the periphery. However, as long as the cells of origin within the ganglion are intact, a new nerve fiber will begin to grow downward and conduction within the nerve may be expected from a few months to two years. Injection of the Gasserian ganglion is more difficult, but it may be followed by relief of pain for a longer time. The most logical indication for an alcohol injection exists in a very old, debilitated individual whose pain originates in and is limited to the mandibular division. It must be realized that injections are not devoid of danger; we have examined patients with paralysis of the oculomotor and facial nerves and with loss of hearing following this type of treatment. Even an extensive experience with injections does not guarantee success for the injector because of the wide variation in skulls. As a general rule the frequency of alcohol injections for the relief of trigeminal neuralgia varies directly with the technical surgical difficulties which the individual surgeon associates with section of the sensory root

At present, surgical approach to the Gasserian ganglion and sensory root is made through the temporal bone at the level suggested by Cushing and Lexer. However, the incision is a longitudinal one which extends upward for about 3 inches from the preglenoid tubercle on the zygoma (Adson). A simple trephine opening is made through the squamous portion of the temporal bone, and this is enlarged with bone-biting forceps. The method of making a large flap of muscle, periosteum, and bone is quite unnecessary. The operative opening

[1] Schloesser, K. Zur Behandlung der Neuralgien durch Alkoholeinspritzungen. Berlin. Klin. Wchnschr., 43, 82, 1906

is enlarged downward as far as possible rather than upward, since the field is better exposed thereby and less retraction of the temporal lobe of the cerebrum is necessary. Upon raising the dura mater by wet cotton sponge dissection from the floor of the middle cranial fossa, it may become necessary to identify several landmarks. These are (1) the foramen spinosum through which the middle meningeal artery enters the cranial cavity; (2) the foramen ovale through which the mandibular division exits from the skull; and (3) the Gasserian ganglion with its sensory root. The entire operation may be performed under local anesthesia, or when the ganglion is exposed, light sodium pentothal anesthesia is induced until the root is severed. However, the patient is awake before leaving the operating room and is relieved immediately of the pain. The sensory loss is for pin prick, cotton wool touch, and temperature stimuli. Deep pressure sense in the face is conserved in the facial nerve.

This operation has been described in some detail because, judging from the advice which patients receive from their doctors, the nature of the procedure and its operative risks are not well understood. Patients have been told that their face would be paralyzed, that they would be mentally unsound after such an operation, and most commonly that the mortality rate is higher than 50 per cent.

We have had 1 death in our own series of patients, an operative mortality of less than 0.18 per cent, and that is about the average for all experienced neurological surgeons.

Recent refinements in technique proposed by Frazier,[1] Stookey,[2] and others have made it possible to perform a subtotal section of the sensory root fibers. If the pain has been limited to the maxillary and mandibular divisions, it may seem advisable to conserve the ophthalmic portion of the root.

As Davis and Haven[3] have shown, since the fibers of the root rotate in their course from the brain stem to the ganglion, and anastomose freely at the hilus of the ganglion, it is necessary to exercise care as to the exact point at which subtotal section is performed. Otherwise, fibers may be conserved in which pain has been present and relief may not be so successful as is possible.

Another nicety in technique is that which conserves the motor root. This structure may be identified easily, and if the superior-medial edge of the ganglion is drawn downward and laterally as Adson has suggested, the motor root will separate from the overlying sensory root quite easily. (Fig. 115.)

[1] Frazier, C. H.: Refinement in the Radical Treatment of Trigeminal Neuralgia, Jour. Am. Med. Assn., 76, 107, 1921.

[2] Stookey, B.: Differential Section of Trigeminal Root in Surgical Treatment of Trigeminal Neuralgia, Ann. Surg., 87, 172, 1928.

[3] Davis, L., and Haven, H.: Surgical Anatomy of Sensory Root of Trigeminal Nerve, Arch. Neurol. and Psychiat., 29, 1, 1933.

Another approach to the sensory root by a suboccipital craniotomy has been suggested by Dandy[1] on the basis that section of approximately half of the sensory root near the pons results in complete relief of pain with but slight sensory changes in the face and that this operation very often discloses small tumor masses or aberrant blood-vessels which are etiological factors in production of the pain. Experimentally and clinically, it has been shown that the sensory loss from the division of the root near the pons and near the ganglion *is the same. Further, the possible surgical accidents* which may occur to the important cranial nerves in the posterior fossa which must be exposed by a suboccipital approach is an important matter not to be regarded lightly particularly since several serious postoperative complications of irreparable dam-

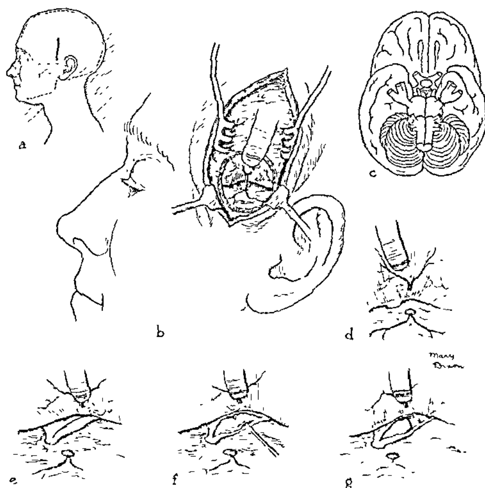


FIG 115 —(*a*) Illustrating the line of the skin incision for trigeminal neurotomy; (*b*) the dura is raised from the floor of the middle fossa until the middle meningeal artery is exposed in the foramen spinosum, (*c*) diagram to show the point at which the posterior root of the trigeminal nerve is severed, (*d*) the middle meningeal artery is ligated and the mandibular division of the trigeminal nerve is exposed; (*e*) the dura mater of the temporal lobe is raised from the dural envelop which encloses the Gasserian ganglion (an incision is made in the envelop), (*f*) a right-angled knife severs the posterior root between the ganglion and the pons, (*g*) the peripheral cut end of the posterior root may be seen after division

[1] Dandy, W E Treatment of Trigeminal Neuralgia by *Cerebellar Route*, Ann. Surg., *96*, *787*, *1932*

age to the cerebellum have been observed following this operation by various neurological surgeons over the country.

Certain conditions may be present following an operation for trigeminal neuralgia which must be explained clearly beforehand to the patient, but none of these approaches in gravity the excruciating pain from which the patient suffers. In the order of their importance, these conditions are: (1) lesions of the cornea, (2) facial paresis, (3) paresthesias, (4) difficulty in mastication, and (5) a feeling of fullness in the ear.

Involvement of the corneal epithelium occurs as the result of a direct injury to the insensitive cornea. It is necessary, therefore, for the patient to exercise more than ordinary caution in the care of his eye. He should wear glasses or goggles when exposed to a strong wind or to dust and cinders. Gauze or cotton should not be used to remove foreign material from the eye because of the danger of injuring the corneal epithelium. The patient must be instructed to irrigate the eye, using either an eye dropper or an eye cup, with physiological saline solution or sterile water twice daily, and to close his eyelids more frequently than he has been accustomed to in order to lubricate the cornea with the normal conjunctival secretions. Boric acid solution and other eye washes irritate the eyes of these patients. While in the hospital, the patient is instructed carefully in the care of his eye and is made conscious of the fact that he must protect his cornea from gross trauma. Dressings are not used in an effort to keep the eyelid closed. They are difficult to hold in place and add a definite hazard to the safety of the cornea. If the cornea becomes ulcerated, the eye may be closed by suturing the eyelids together until the cornea is healed. However, if the patient is instructed to report the moment the eye becomes red and if he is then told to keep the eyelids closed voluntarily, to irrigate the eye, and to continue this simple treatment until all redness has disappeared, usually within forty-eight hours all signs of inflammation have disappeared.

Paresis of the facial muscles may follow division of the sensory root of the fifth nerve, and all of the facial muscles on the affected side are involved. It occurs in about 2 per cent of the cases and may be present immediately after or it may appear some days following operation. Its occurrence has been ascribed to trauma within the pons produced by avulsion of the sensory root, to the slow oozing of incompletely controlled bleeding, and to traction upon the greater superficial petrosal nerve, a branch of the geniculate ganglion of the facial nerve. The latter is the most likely and accurate explanation since this nerve crosses the petrous portion of the temporal bone and may be injured as the dura mater is raised from the floor of the middle

lesion may be verified as an aneurysm of the internal carotid artery either at autopsy or at operation; or there may be a small meningioma, wholly confined to the dural envelop which encloses the Gasserian ganglion. Not infrequently tumors of the nasopharynx grow upwards into the floor of the skull and involve the paratrigeminal area.

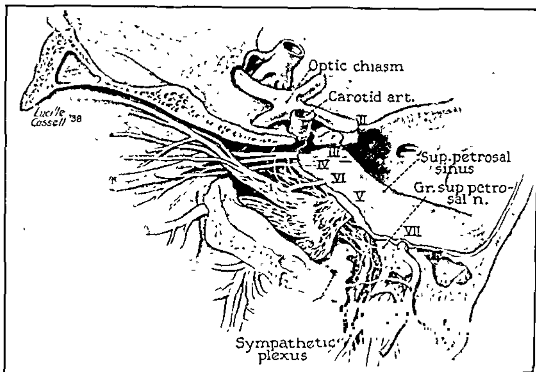


Fig. 116.—Drawing of the anatomical relations of the paratrigeminal area showing the cranial nerves indicated by Roman numerals; the Gasserian ganglion with its three divisions 1, ophthalmic; 2, maxillary; 3, mandibular.

Without exception, all of these patients presented themselves for relief of the severe pain which they suffered in the area of distribution of the trigeminal nerve. Failure to obtain relief from this persistent excruciating pain by the usual analgesic drugs or by alcohol injections had led them to seek surgical attention. This pain is one of the first symptoms noted by the patient and in our experience has occurred in one, two or all branches of the Gasserian ganglion, but without fail the ophthalmic division has always been involved. A trigger zone so pathognomonic of trigeminal neuralgia may or may not be present. Though sharp and severe, the pain seldom has had the paroxysmal character of true trigeminal neuralgia, nor is it as shocking and terrifying to the patient. Often the pain seems to radiate peripherally from an origin located indefinitely by the patient as "somewhere behind the eye." Several patients have complained

of a severe, dull headache localized to a point deep within the anterior portion of the cranium on the affected side.

Very often we have found that when this syndrome has been due to a small meningioma, originating within the dural envelop which encloses the Gasserian ganglion, there have been small patchy areas of paresthesia, numbness and even complete loss of sensation to pin prick and touch stimuli in the ophthalmic and maxillary areas of the face on the side of the lesion. This has been so constant an occurrence, if examined for carefully and meticulously, that we have come to regard it as pathognomonic of the presence of such a tumor.

Accompanying these symptoms of involvement of the trigeminal nerve have been the signs produced by involvement of the third, fourth and sixth cranial nerves, which innervate the extra-ocular muscles. As might be expected, these nerves may be affected singly or in any possible combination. The most common manifestations have been those which pointed to the oculomotor nerve and have been characterized by a dilated pupil, an actual ptosis of the upper eyelid and divergence of the eyeball to the outer canthus. In two instances, in which there was a meningioma, there was a complete paralysis of the external rectus muscle, pointing to a lesion of the abducens nerve, and in another case, in which there was a large aneurysm of the internal carotid artery, a complete opthalmoplegia was present. In no instance in which a meningioma was found and removed at operation have the extra-ocular muscles failed to recover their function. In fact, the marked ocular symptoms have been more characteristic of the cases in which there were aneurysms of the internal carotid artery.

When the sympathetic nerve fibers which surround the carotid artery and accompany the ophthalmic division of the trigeminal nerve are involved, a typical Horner's syndrome is produced with enophthalmos, a narrowed palepbral fissure and a small pupil which will fail to dilate after the introduction of cocaine solution. Under these circumstances, which most often occur with aneurysms of the internal carotid artery, the ciliospinal reflex is also absent.

That surgical help can be given to at least one-half of these patients in our experiencè and that a shrewd guess can be made that a small meningioma rather than an aneurysm is present, and that these patients come to the surgeon for relief of the severe pain in the face is illustrated by the following case:

A woman aged fifty years first complained of a jabbing, knife-like pain in the left eye in June, 1937. This was not present constantly but came in attacks and soon involved the left frontal and the left maxillary areas. Cold air, rubbing the left temporal area or touching the site of the removed left canine tooth brought on the pain. At frequent intervals diplopia would be present for hours or days at a time.

On examination, stimulation of the zones described by the patient produced a sharp increase in her pain but it was obvious that she was never entirely free from a pain described as "deep in the left eye" No impairment of the extra-ocular muscle movements could be elicited on examination. There was a loss of sensation to light touch and pin prick stimuli over the left upper

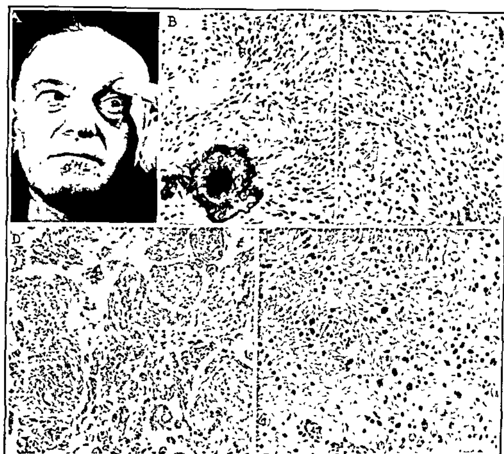


FIG. 117.—Lesions of the paratrigeminal area. (*A*) Photograph of patient which shows ptosis and complete ophthalmoplegia on the left; (*B*) photomicrograph of the nasopharyngeal tumor which was a myxo-chondro-sarcoma, × 130; (*C*) photomicrograph of intracranial extension of the tumor which shows the same structure as in (*B*), × 130, (*D*) photomicrograph of tumor of the Gasserian ganglion, × 80, which shows large and small nests of meningiomatous cells with many small whorls in a pre-psammomatous stage, (*E*) photomicrograph of tumor of the Gasserian ganglion, × 240, which shows atypical ganglion cells and a predominance of cells with small, darkly stained nuclei in a matrix of dense fibrous tissue.

lip, left ala and left side of the nose which extended laterally about 2 cms. The left upper and lower eyelids were also insensitive

At operation the Gasserian ganglion enclosed in its dural envelop appeared coarse, large and red When the envelop was opened a soft purple mass was found occupying the site of the ganglion and wholly enclosed within its dural envelop The mass was removed completely and the sensory root was divided.

The patient has had no recurrence of her pain and no new symptoms have developed during the three years following her operation.

These tumors are not tumors of the Gasserian ganglion and show none of the microscopic characteristics of such tumors, which are not uncommonly found. The tissue which comprises the intracranial extension of a nasopharyngeal tumor often may be composed of a uniform, epithelioid type of cells with central round, granular or reticular nuclei, which are arranged in poorly defined whorls about central blood-vessels. Other more elongated endothelial-like cells are present and mistoses are fairly common. (Fig. 117.)

The story of the patients with an aneurysm which produces paratrigeminal symptoms and signs is not a happy one but in view of the potentialities of the lesion it may be of much longer duration than one would at first imagine:

A woman aged forty-seven years had a severe pain in the right eye followed by a terrific generalized headache while attending a funeral in 1932. Her vision was dim and she had a diplopia The headache, present since the onset, was described as a sharp, continuous pain over the right temporal area which varied in intensity. This pain seemed to "shoot through" the right eye in attacks and when this occurred, as it did about once a week, she became nauseated, vomited and had a true vertigo. A "boiling, drawing" pain had been present over the vertex of the skull since 1934. Although she had diplopia from the onset, she did not notice that her right eye was turning to the left and that the pupil was large until a year later.

In 1937 it was noted that the right upper eyelid was "drooping" and she could not open her eye easily. Simultaneously a tingling, fiery, sharp pain occurred frequently in the right side of the face and would last about fifteen minutes, to be followed by numbness in the right side of the tongue, the teeth and gums on the right side.

There was a definite ptosis of the right upper eyelid, and the right pupil was larger than the left and reacted sluggishly to light. The visual fields and acuity were normal in both eyes. In looking upward and to the left and right, the right eye did not move at all but did move on looking down. There was diminution to pin prick and touch stimuli over the right maxillary area and on the right side of the tongue.

At operation an enormous aneurysm of the internal carotid artery extended upward and laterally into the middle fossa.

Extensive paralysis of the extra-ocular muscles has been quite characteristic of the cases of aneurysm of the internal carotid artery and in our experience the oculomotor nerve has been more constantly involved than the other nerves to the extra-ocular muscles. It is our opinion that in the cases in which there is an aneurysm, the character and distribution of the pain does not resemble the pain of trigeminal neuralgia as closely as it does in the cases in which there are meningiomas of the dural envelope of the ganglion. Neither have we found the presence of a sensory loss as constant in the instances of aneurysm.

FACIAL PARALYSIS

"The muscles of the cheek on the left side are wasted, and there appears to remain nothing but the thin integuments which hang upon the side of the face, as if dead, without having any action in them, or wrinkles, as in the right cheek; and when he speaks, this cheek is alternately puffed out and then collapsed; the air first distending it, as it were a bag, and then escaping at the angle of the mouth. His whole mouth is drawn to the right side, thus producing most remarkable distortion of the face. Whatever action there is in the mouth is altogether owing to the contraction of the muscles on the right side of it; the left angle hangs loose and is quite passive; and the saliva is allowed to flow constantly out upon the lower lip on this side."

By this brilliant description of the appearance of a patient whose facial nerve had been severed, when he had been "tossed by a bull, the horn going in at the angle of the jaw," Sir Charles Bell attempted to convince his colleagues that the facial nerve alone was responsible for "the remarkable distortion of the whole face."[1] For years, Bell had held the conviction that the nerves, and especially those of the face, had distinct functions. His notebooks became filled with cases which supported his view, but there was a time "in which I would have given all that I was worth to have such proofs." Even with the evidence and facts before them, Bell complained of "certain men, high in science, and respectable in our profession, who have denied them with a heat and pertinacity which I can never understand, and which surely ought not to belong to such an inquiry."

Bell described many cases of facial paralysis due to many different etiological factors, but at present the term "Bell's palsy" has been reserved, by more or less common usage, for those cases in which the facial weakness occurs rather abruptly unassociated with recognizable ear disease or direct trauma. It is quite generally conceived to be due to swelling of the nerve within the facial canal with the production of a complete physiological interruption of the nerve fibers

The facial nerve is the cranial nerve most frequently involved by trauma or disease and the one for which surgical repair is most often indicated It may be severed by a stab or gunshot wound, or it may be injured inadvertently during a mastoid operation as was true in most of our patients. The nerve may be torn within the cranial cavity by a fracture of the base of the skull, and cases are on record in which fragments of bone have compressed the nerve within the facial canal Facial paralysis may accompany an otitis media, or it may follow some time after a mastoidectomy in which event it may be assumed that compression and not anatomical severance has

[1] Bell, Charles The Nervous System of the Human Body, Embracing the Papers Delivered to the Royal Society on the Subject of the Nerves, p 135, 1833.

produced the paralysis. Bilateral facial paralysis is not a common condition, but we have observed one case in which both facial nerves were injured when the head of the patient was pinned between a concrete highway and the motor of an overturned automobile. Frequently the facial nerve may be intimately surrounded by the growth of a tumor of the parotid gland or, as in the first case we operated on, by a tumor situated between the angle of the jaw and the mastoid bone.

As Bell so eloquently described it, paralysis of the trunk of the seventh cranial nerve is characterized by the complete absence of movements of expression on the paralyzed side, with inability to wrinkle the forehead or to close the eye. Attempts to close the eye are associated with a movement of the eyeball upward, a symptom known as Bell's phenomenon. The patient is unable to pucker his lips, to smile or show the teeth on the paralyzed side. Because of atony, the expression is "washed out," the angle of the mouth droops, the nasolabial fold is obliterated, and there is an effacement of wrinkles on the forehead. All of these signs are exaggerated in any voluntary movements of the face or during emotional expressions. If the nerve is injured within the facial canal before its exit from the stylomastoid foramen, taste is lost over the anterior two-thirds of the tongue. Frequently, only a partial injury may occur, and in addition to the preservation of reaction of the muscles to faradic stimulation more than fourteen days after injury, certain muscle functions remain to indicate recoverability of the lesion. Not only may the eyelids of the affected side be closed more completely than in the case of total paralysis, but twitches may be observed in the lower lid accompanying the winking of the opposite eyelids consequent to threatening gestures. The nasolabial fold is not completely obliterated, and the lips on the affected side are not separated so markedly. In contrast to these signs and symptoms facial paralysis, which accompanies a lesion of the facial nerve fibers from the cortical cells in the motor cortex of the cerebrum to the facial nerve nucleus in the pons, shows no involvement of the muscles of the upper portion of the face. The frontalis muscle is wrinkled equally well on both sides, and although the palpebral fissures may be strikingly different in size, the patient is able voluntarily to close the upper eyelid though it be weakly performed.

Those cases of facial paralysis which occur as the result of an inflammatory or toxic lesion usually recover spontaneously over a time varying from a few days to a few months. A surgical problem is not presented by these patients, but we are accustomed to treat them by massage of the facial muscles, by galvanic stimulation, and by a simple type of adhesive splint which prevents the paralyzed

muscles from stretching. The large group of cases in which facial paralysis is complete and permanent is the important entity which is deserving of surgical consideration. In addition to the physical handicap which is present, the question of personal appearance is a prominent one. These patients are deprived of the power of expressing their personality by modification of the facial muscles; the emotions are but half revealed, and there is a dreadful handicap in any activity, commercial or social, in which personal appearance is of importance.

Surgical methods for the repair of a facial nerve paralysis fall into two groups; those in which the nerve defect is assumed to be irreparable and attempts are made to repair the defective facial musculature by the transplantation of fascia, and those in which an attempt is made to innervate all of the facial muscles by means of nerve anastomosis. As a matter of fact, both methods may be used to the distinct advantage of the same patient. As elsewhere in peripheral nerve surgery, the ideal form of treatment is an end-to-end suture of the severed nerve, and although this is a difficult and serious procedure in its course within the facial canal, the nerve can be sutured quite readily in its course from the stylomastoid foramen to the point of its division into its many branches. However, in the majority of cases the patient presents himself for repair of his deformity long after the original injury has occurred, and when scar tissue and callus have developed within the facial canal so that a direct end-to-end suture is impossible. Consequently, other methods of nerve repair must be employed. Frequent attempts have been made to correct a facial paralysis by the anastomosis of the distal end of the facial nerve beyond the stylomastoid foramen with the central end of another cranial nerve. (Fig. 118.)

The spinal accessory and the hypoglossal nerves have been most frequently used in these operations. Faure and Furet, in 1898, first performed the operation of anastomosing the spinal accessory and the facial, and Korte, in 1901, first used the hypoglossal nerve in such a nerve crossing.

Many experimental investigations have been carried out to study the results of such anastomoses, and many clinical cases have been operated upon. In 1911 and later Kennedy reported that approximately equal results may be obtained with either the hypoglossal or spinal accessory nerves. In Cushing's case of spino-facial suture, there were marked movements of the facial muscles associated with turning the head or raising the shoulder Ballance and Stewart have stated that the hypoglossal-facial suture was preferable to spinal accessory-facial crossing because of the fact that the cortical centers for the face and tongue are closer together The shoulder and facial muscle movements are not synergistic and have little or no association.

Another problem always is presented in the consideration of the operation of nerve crossing. This concerns the paralysis which results from section of the substituted nerve. Use of the hypoglossal

nerve results in an atrophy of the corresponding half of the tongue, while section of the spinal accessory is followed by impairment of function of the arm and shoulder. The choice between these two conditions depends a great deal upon the occupation of the patient

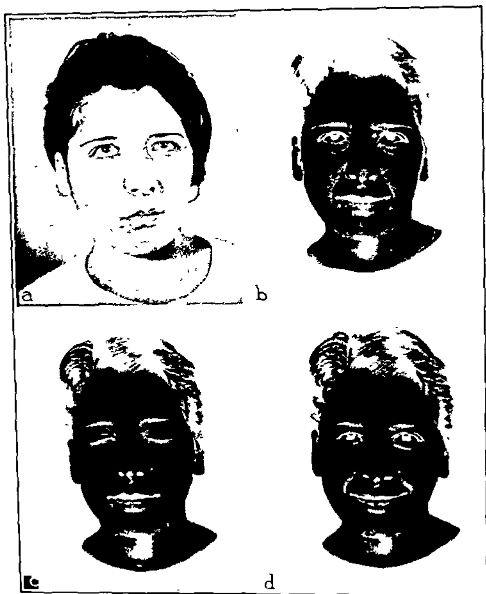


Fig. 118.—(*a*) Facial paralysis due to a gunshot wound; (*b*, *c* and *d*) three years following anastomosis of the facial and hypoglossal nerves.

although we have now come to regard this as a minor consideration since in our hands, at least, the most successful results have been obtained after hypoglossal-facial suture, and none of our patients has complained of any discomfort produced by atrophy of the tongue.

We have performed a spinal accessory-facial anastomosis upon 1 patient and a hypoglossal-facial anastomosis upon 14 others. In the first case, the patient had a complete facial paralysis on the left side which was due to a walnut-sized tumor mass between the mastoid process and the angle of the jaw. This had been present for a considerable time, but her face had been paralyzed for two years. At operation, the mass was removed, but it completely surrounded the facial nerve and infiltrated it near the mastoid foramen so that it was necessary to section the nerve. The return of tone in the facial muscles was quite rapid after the anastomosis so that the symmetry of her face became more nearly normal. One year after operation she was unable to close her eyelid although she had good voluntary movements about her mouth which were unassociated with movements of the shoulder. The most successful results were obtained by use of the hypoglossal nerve in 11 cases in which the facial nerve was injured during a mastoidectomy, in one in which it was injured within the canal by a bullet wound; and in another case a skull fracture damaged the nerve. In each of these cases muscle tone and the symmetry of the face returned quickly. Movements of the face then became possible, at first associated with movements of the tongue. It has taken each of our patients about two years to dissociate their movements so that voluntary movements of the facial muscles were possible without movements of the tongue. Emotional expressions approximating the normal have occurred last of all, as has the voluntary movement of closing the eyelid. (Fig. 119.)

We believe that the postoperative treatment is as important as is the meticulous detail with which the actual suture of the nerve ends is carried out. The facial muscles should be kept from sagging and overstretching by a simple mechanical device which will lift the angle of the mouth and the muscles of the cheek. An adhesive tape bridge with one end attached to the skin in the anterior temporal region and the other to the skin near the angle of the mouth is very efficient The patient should also receive daily massage and gentle galvanic or sinusoidal stimulation to the facial muscles. Educative exercises should be taught to the patient, and these should be performed before a mirror, particularly after the appearance of the first return of function. The fundamentals of the operation and the action of the nerve-muscle relationship should be explained to the patient, and it should be pointed out to him that future improvement lies to a great extent with himself. One of our patients has an excellent return of expressional movements, but when asked to perform certain voluntary acts finds difficulty in dissociating the movement of her tongue. She has, however, devised exercises which have resulted in a marked improvement.

Many ingenious methods have been devised to relieve the disfiguring asymmetry of the face by elevation of the lower eyelid or the angle of the mouth with pedunculated muscle flaps, aluminum bronze wire and fascia lata transplants. Blair's method makes use of several fascia lata transplants which are anchored in the temporal fascia and are attached at the angle of the mouth, the nasolabial fold and the upper and lower lips. This method may be used to



Fig. 119 —Result of facial-spinal accessory anastomosis: (*a*) Before operation; (*b*) nine months after operation; (*c*) four years after operation; (*d*) five years after operation (Courtesy of Pollock and Davis, *Peripheral Nerve Injuries*)

great advantage to restore facial asymmetry and to prevent overstretching of the paralyzed muscles while waiting for nerve regeneration and the return of muscle function to occur.

The remarkable improvement in appearance and morale of these unfortunate patients leaves no doubt whatever in our minds that

every possible effort should be made to aid them in returning to their former social and economic positions in life.

Hemifacial Spasm.—Patients with spasm of the facial muscles on one side of the face are a particularly unhappy group. They have been told that they are psychoneurotic because no cause for the spasm can be found and because they may be free from the spasm for considerable intervals during which the facial muscles are apparently normal. However, surgical exposure and deompression of the facial nerve within the facial canal has been helpful in a small group of Woltman's patients. Also, Campbell and Keedy[1] have recorded a cirsoid aneurysm of the basilar artery in 2 patients with hemifacial spasm. It is to be hoped that these patients may be relieved by carefully planned surgical procedures.

AURAL VERTIGO

Recently, the eighth cranial nerve has come in for its share of surgical attention in the relief of a group of symptoms which have been classified as Ménière's disease. In 1861, Ménière[2] presented 2 patients before the French Academy of Medicine who suffered from sudden attacks of vertigo accompanied by nausea and vomiting. As they walked, both patients vividly described a sensation of being forcibly pulled to one side or the other. Interestingly enough, Ménière described the condition as "Diseases of the Internal Ear Presenting the Symptoms of Apoplectiform Cerebral Congestion," but emphasized strongly the fact that neither the cerebrum or cerebellum were actually involved in the production of the symptoms. In the first patient there had been evidence of a hemorrhage within the inner ear, and since then a pathological condition of the labyrinth has been accepted as the basic etiological factor, without adequate proof.

It is obvious that hemorrhage into the labyrinth may not be the only pathological condition responsible for symptoms of what might better be termed "aural vertigo." Many purely hypothetical pathological lesions have been suggested as the cause of the disturbing symptoms complained of by these patients, and what would appear to be logical treatment has been instituted without any effect. For example, it has been supposed that there was an increased secretion of cerebrospinal fluid and repeated spinal punctures have been performed; stimulation, or inhibition, of the sympathetic and parasympathetic nervous system have been treated by the appro-

[1] Campbell, E., and Keedy, C. Hemifacial Spasm A Note on the Etiology in Two Cases. Jour. Neurosurg., 4, 342, 1947.

[2] Ménière, W. P. Maladies de l'oreille interne offrant les symptomes de la congestion cerebrale apoplectiforme, Gaz. méd. de Paris, 16, 88, 1861.

priate drugs which have been proven to be inhibitors or stimulators of these fibers.

In 1938, Hallpike and Cairns[1] examined the temporal bones of 2 patients with aural vertigo and described gross distention of the endolymphatic system with degenerative changes in the sensory elements. Others have confirmed these findings and it might be inferred that the lesion, therefore, is in the inner ear. Dandy[2] insisted that the lesion was along the afferent pathway, probably in the eighth cranial (acoustic) nerve. No striking pathological changes have been found in the nerve but it has been suggested that anomalous vessels may press upon and irritate it. Certainly, acoustic nerve tumors or aneurysms of the vertebral artery may produce symptoms similar to aural vertigo without the paroxysmal attacks which are so characteristic. An alteration in capillary permeability in the capillaries of the stria vascularis in the labyrinth has been suggested as a basic fault and receives support from the histological examinations of the endolymphatic spaces and the benefits which follow various forms of medical treatment. However, some organic changes must occur to account for the loss of hearing and response to vestibular tests.

Regardless of what may be established as the etiology or pathology, patients with aural vertigo suffer from paroxysmal, explosive, terrific attacks of vertigo which are accompanied by nausea and vomiting. In an attack, the patient may fall to the floor violently and be unable to arise, or he may deviate forcibly and suddenly to one side or another as if pulled there by a large magnet. There is deafness and tinnitus on the affected side and even a contralateral nerve deafness may be present. The attack, which certainly bears a close resemblance in its explosive character to trigeminal neuralgia, may be preceded by a definite aura of fullness in the ear or a feeling of warmth on the back or side of the neck. Also like trigeminal neuralgia, there may be long intervals between attacks during which the patient is well except for the persistence of unilateral tinnitus and a gradual increase in deafness.

In some patients tinnitus, deafness and vertigo have a simultaneous onset which makes recognition of the disease relatively easy but in many any one of these symptoms may antedate the other by many months or years. Sometimes there is a bilateral tinnitus but deafness is more marked in one ear than the other, or one labyrinth is more easily stimulated than the other, or gives no response at all. It is not uncommon to find that in those patients whose attacks are not so

[1] Hallpike, C. S., and Cairns, H. C.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol., 53, 625, 1938.

[2] Dandy, W. E.: Ménière's Disease; Its Diagnosis and a Method of Treatment, Arch. Surg., 16, 1127, 1928.

every possible effort should be made to aid them in returning to their former social and economic positions in life.

Hemifacial Spasm.—Patients with spasm of the facial muscles on one side of the face are a particularly unhappy group. They have been told that they are psychoneurotic because no cause for the spasm can be found and because they may be free from the spasm for considerable intervals during which the facial muscles are apparently normal. However, surgical exposure and deompression of the facial nerve within the facial canal has been helpful in a small group of Woltman's patients. Also, Campbell and Keedy[1] have recorded a cirsoid aneurysm of the basilar artery in 2 patients with hemifacial spasm. It is to be hoped that these patients may be relieved by carefully planned surgical procedures.

AURAL VERTIGO

Recently, the eighth cranial nerve has come in for its share of surgical attention in the relief of a group of symptoms which have been classified as Ménière's disease. In 1861, Ménière[2] presented 2 patients before the French Academy of Medicine who suffered from sudden attacks of vertigo accompanied by nausea and vomiting. As they walked, both patients vividly described a sensation of being forcibly pulled to one side or the other. Interestingly enough, Ménière described the condition as "Diseases of the Internal Ear Presenting the Symptoms of Apoplectiform Cerebral Congestion," but emphasized strongly the fact that neither the cerebrum or cerebellum were actually involved in the production of the symptoms. In the first patient there had been evidence of a hemorrhage within the inner ear, and since then a pathological condition of the labyrinth has been accepted as the basic etiological factor, without adequate proof.

It is obvious that hemorrhage into the labyrinth may not be the only pathological condition responsible for symptoms of what might better be termed "aural vertigo." Many purely hypothetical pathological lesions have been suggested as the cause of the disturbing symptoms complained of by these patients, and what would appear to be logical treatment has been instituted without any effect. For example, it has been supposed that there was an increased secretion of cerebrospinal fluid and repeated spinal punctures have been performed; stimulation, or inhibition, of the sympathetic and parasympathetic nervous system have been treated by the appro-

[1] Campbell, E., and Keedy, C.. Hemifacial Spasm. A Note on the Etiology in Two Cases, Jour Neurosurg., 4, 342, 1947

[2] Ménière, W P Maladies de l'oreille interne offrant les symptomes de la congestion cerebrale apoplectiforme, Gaz. méd. de Paris, 16, 88, 1861.

priate drugs which have been proven to be inhibitors or stimulators of these fibers.

In 1938, Hallpike and Cairns[1] examined the temporal bones of 2 patients with aural vertigo and described gross distention of the endolymphatic system with degenerative changes in the sensory elements. Others have confirmed these findings and it might be inferred that the lesion, therefore, is in the inner ear. Dandy[2] insisted that the lesion was along the afferent pathway, probably in the eighth cranial (acoustic) nerve. No striking pathological changes have been found in the nerve but it has been suggested that anomalous vessels may press upon and irritate it. Certainly, acoustic nerve tumors or aneurysms of the vertebral artery may produce symptoms similar to aural vertigo without the paroxysmal attacks which are so characteristic. An alteration in capillary permeability in the capillaries of the stria vascularis in the labyrinth has been suggested as a basic fault and receives support from the histological examinations of the endolymphatic spaces and the benefits which follow various forms of medical treatment. However, some organic changes must occur to account for the loss of hearing and response to vestibular tests.

Regardless of what may be established as the etiology or pathology, patients with aural vertigo suffer from paroxysmal, explosive, terrific attacks of vertigo which are accompanied by nausea and vomiting. In an attack, the patient may fall to the floor violently and be unable to arise, or he may deviate forcibly and suddenly to one side or another as if pulled there by a large magnet. There is deafness and tinnitus on the affected side and even a contralateral nerve deafness may be present. The attack, which certainly bears a close resemblance in its explosive character to trigeminal neuralgia, may be preceded by a definite aura of fullness in the ear or a feeling of warmth on the back or side of the neck. Also like trigeminal neuralgia, there may be long intervals between attacks during which the patient is well except for the persistence of unilateral tinnitus and a gradual increase in deafness.

In some patients tinnitus, deafness and vertigo have a simultaneous onset which makes recognition of the disease relatively easy but in many any one of these symptoms may antedate the other by many months or years. Sometimes there is a bilateral tinnitus but deafness is more marked in one ear than the other, or one labyrinth is more easily stimulated than the other, or gives no response at all. It is not uncommon to find that in those patients whose attacks are not so

[1] Hallpike, C. S., and Cairns, H. C.: Observations on the Pathology of Ménière's Syndrome, J Laryng & Otol, 53, 625, 1938

[2] Dandy, W. E.: Ménière's Disease; Its Diagnosis and a Method of Treatment, Arch Surg., 16, 1127, 1928.

disabling, when a complete loss of hearing occurs, the attacks of vertigo disappear. While tinnitus usually means a ringing, it does include sounds described as buzzing, swishing, sizzling, whistling, drumming and beating.

Often a typical attack can be initiated by performing a caloric test on the affected side but this is not always the case. Sometimes, the direction toward which the patient feels pulled; the character of the nystagmus which may occur and the absence of, or difference in, hearing are helpful in making the diagnosis. In our experience, tumors of the acoustic nerve have never produced the violent vertigo from which these patients suffer. Tumors of the cerebello-pontile angle, and in particular, those which involve the dorsal surface of the cerebellar hemisphere may produce vertigo and nystagmus which are similar.

The majority of patients with aural vertigo have paroxysmal attacks in which the symptoms are mild and sedatives or dramamine will give them prompt relief. The attacks may in time subside spontaneously or can be prevented by a careful regime of a salt-free diet, dehydration and histamine. By a series of clinical experiments, Furstenberg[1] came to the conclusion that these disabling attacks of vertigo are the result of the fact that the local tissues involved have either an increased avidity for, or an unusual sensitivity to sodium. By placing the patient upon a salt-free diet and administering ammonium chloride (3.0 gm in a capsule with each meal for three successive days and an interval of two days without this drug), he has been able to relieve his patients completely.

Horton[2] has been able to stop the acute attack of vertigo, nausea and vomiting by the intravenous injection of histamine base. Histamine diphosphate, (2.75 mg., in 250 cc. of physiological saline solution), may be given intravenously at the rate of 50 to 60 drops per minute and can be repeated on successive days. To prevent future attacks, Horton gives his patients 0.1 to 0.2 mg. of histamine subcutaneously two to four times a week but there is some question as to its efficacy. Dramamine (50 mg. tablets by mouth) are helpful in stopping the acute attack of vertigo.

It is our custom to place these patients upon medical treatment according to Furstenberg's method for a time sufficient to determine definitely whether or not they are improved. We have observed patients whom we believe suffered from true attacks of aural vertigo and in whom we can find no other organic cause for their symptoms. In some of these the attacks have been severe and disabling and medi-

[1] Furstenberg, A. C. Ménière's Symptom-Complex, Medical Treatment. Trans. Am. Otol. Soc., 24, 126, 1934.

[2] Horton, B. T. The Use of Histamine in *Ménière's Disease*, *Surg., Gynec. and Obst.*, 72, 417, 1941.

cal treatment has failed, so that surgical section of the eighth cranial nerve has been performed. In the remainder, the attacks have disappeared or continue to improve upon medical treatment.

Irrespective of the etiology or pathology, these patients can be relieved by severance of the eighth cranial nerve. McKenzie[1] has stated that section of the vestibular portion of the acoustic nerve alone is feasible, thus leaving the cochlear portion intact. There is usually a gross partition between the two divisions, but microscopically there is an intermingling of vestibular and cochlear fibers along this apparent line of cleavage. Therefore, the nerve cannot be split accurately in a microscopic sense but this is possible for all clinical purposes.

All of our patients operated upon have been relieved of their attacks with the exception of one who had a recurrence five months after operation and in whom the anterior inferior cerebellar artery had to be dissected from the nerve before a partial section of the nerve was made. McKenzie has found a definite relation between the nerve and this artery in 5 patients; in fact, in 1 case the artery perforated the acoustic nerve; and he is of the opinion that this may be the etiological factor in many patients, especially those with arteriosclerosis.

Finally, we have observed several patients who may possibly belong to Dandy's group of "pseudo-Ménière's disease," who have been relieved of their symptoms by the most conservative and elementary procedures carried out by their otologist. These patients are easily upset by the motion of a hammock or swing; they become car sick, train sick, or sea sick very promptly. Each patient must be studied carefully and in our opinion, treated conservatively until it is definitely established that he cannot be relieved by medical treatment or that his attacks are increasing in severity. Then, the patient can be relieved by section of the eighth nerve without any great surgical risk.

GLOSSOPHARYNGEAL NEURALGIA

Glossopharyngeal neuralgia is characterized by the sudden onset of an excruciating, paroxysmal, knife-like pain which shoots from the ear, base of the tongue or posterior pharyngeal wall. Slight twinges of pain may precede the severe major attack which comes quickly and disappears quickly. Drinking cold water, talking, laughing, yawning, sneezing, touching the pinna of the ear, but most often swallowing, may initiate an attack. There are intermissions in the

[1] McKenzie, K. G.: Intracranial Division of the Vestibular Portion of the Auditory Nerve for Ménière's Disease, Canad. Med. Assn. Jour., 34, 369, 1936.

paroxysms of pain and there are remissions in the attacks. Trigger areas or dolorogenic zones are present in this disease in the tonsillar fossa, on the posterior wall of the pharynx or less commonly, on the pinna of the ear. The pain is usually unilateral, but rare cases of bilateral involvement are upon record.

The etiology of the disease is unknown, and no pathological lesions have been found to explain the condition satisfactorily. Whereas, we have encountered 556 patients with trigeminal neuralgia, we have only 6 cases of glossopharyngeal neuralgia in our records

Harris[1] first described glossophrayngeal neuralgia in 1921 although in 1910 Weisenburg and in 1920 Sicard and Robineau had recorded the symptoms without actually being aware of the true nature of the condition. Since then, Doyle,[2] Peet,[3] and others have described the disease and have sectioned the glossophrayngeal nerve for its relief

Harris has given an excellent description of the distribution and radiation of the pain:

"The pain usually starts in the region of the tonsil or the root of the tongue and is apt to be produced by the act of swallowing or yawning, or by a touch on the tonsil or wall of the pharynx; the pain radiates round the pharynx and palate and externally into the region of the auditory meatus, pinna, and the back of the lower jaw In some cases a light touch upon the pinna will start the pain, which may indeed be limited for years to the region of the ear. During the spasms of pain, which may be very frequent, the patient is very liable to a hawking cough, as though attempting to expel some irritant from the throat."

During the attack of severe pain, one of our patients would insert his index finger into the external auditory meatus and violently turn it about in an effort to get relief. In another, saliva drooled from the mouth as the patient sat with his head forward and his chin depressed in an effort to prevent the slightest swallowing movement.

It is obvious that this disease is the counterpart of trigeminal neuralgia and the only differences lie in the distribution of the pain and the location of the trigger areas. In fact, anesthetization of the tonsillar fossa, in which the trigger area often lies in this disease, is followed by relief of pain and serves as a reliable point of differentiation between the two conditions.

The first surgical attempts to relieve this disease were all made extracranially and involved the glossopharyngeal, the vagus, the superior cervical sympathetic ganglion, the pharyngeal branches of the vagus, and even the hypoglossal nerve. Eventually, the present

[1] Harris W Persistent Pain in Lesions of the Peripheral and Central Nervous System, Brain, 44, 557, 1921

[2] Doyle, J B A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol and Psychiat., 9, 34, 1923

[3] Peet, M M Glossopharyngeal Neuralgia, Ann Surg., 101, 256, 1935

successful operation was evolved, which consists of the intracranial section of the ninth cranial nerve alone. This approach is relatively simple, safe, and involves no great danger of injury to the vagus nerve. The results of the operation have been uniformly good.

Until this operation was performed, our knowledge of the actual function of the glossopharyngeal nerve was fragmentary and inaccurate. Vernet had said that a destructive lesion of the ninth cranial nerve caused paralysis of the superior constrictor muscle of the pharynx which resulted in difficulty in swallowing solid food. Like much of the work which followed, his conclusions were based on the study of symptoms produced by tumors or extracranial trauma in the region of the jugular foramen from which the ninth, tenth, and eleventh nerves exit from the skull.

Peet found no demonstrable paralysis of the muscles of the pharynx following this operation and examinatuion of our patients has confirmed this finding. Neither do the patients complain of any difficulty in swallowing solid or liquid food. As Peet pointed out, in some cases there is a considerable area in which overlapping of sensation occurs into the accepted anatomical distribution of the ninth nerve. Usually, therefore, there is a border of diminished sensation about the anesthetic areas which exist at the base of the tongue, in the tonsillar fossa and on the anterior part of the soft palate following section of this nerve. In one of our patients there was no area of complete anesthesia which could be demonstrated. although the gag reflex was lost and sensation on the lateral and posterior pharyngeal walls was considerably reduced. In any event, none of our patients has complained of an impairment of either motor or sensory function following relief of their pain.

LESIONS OF THE LAST FOUR CRANIAL NERVES

The trigeminal, facial, acoustic, and glossopharyngeal are the only cranial nerves upon which a direct surgical attack is commonly made to relieve or repair a pathological process. However, the last four cranial nerves (glossopharyngeal, vagus, spinal accessory, and hypoglossal) are not infrequently involved extracranially. Prior to World War I such cases were reported as due to a tumor or to cervical adenitis, but during the war many instances of direct injury to the lateropharyngeal space were reported, and we have had the opportunity of observing some of these cases which Pollock examined and recorded.

The lateropharyngeal space is bounded above by the base of the skull in the region of the jugular foramen, which is an irregular opening, varying in size and shape, placed between the petrous portion of the temporal bone and

the jugular process of the occipital bone. It is commonly divided into three compartments by spicules of bone which bridge it Through the anterior compartment the inferior petrosal sinus passes; the internal jugular vein and meningeal branches from the occipital and ascending pharyngeal arteries pass through the posterior compartment and between the two are the glossopharyngeal, vagus, and spinal accessory nerves.

The cases which present lesions of all or several of the last four cranial nerves have been described upon the basis of the symptoms which are added to a pure laryngeal hemiplegia. So, in the literature as many syndromes as there are authors of reported cases will be



Fig. 120 —(*a*) Facial weakness and narrowed palpebral fissure, enophthalmos and myosis, (*b*) atrophy of tongue, (*c*) anterior dislocation of shoulder girdle on attempts to shrug shoulder. (Courtesy of Pollock and Davis, Peripheral Nerve Injuries)

found, and only confusion has resulted because the number of syndromes is limited only by the possible combinations of complete or incomplete paralysis and the descriptive abilities of the author.

One of the outstanding features of these cases is, however, that the ninth, tenth, and eleventh cranial nerves are rather consistently injured extracranially together, regardless of what other nerves may be affected. Because of their close relation to the internal carotid artery, internal jugular vein and cervical sympathetic trunk, it seems almost impossible to project the course of a wound which would injure these nerves and leave the artery and vein intact. However, it is possible for a bullet to pass obliquely from the mastoid region on one side to the malar bone on the other and produce an extracranial lesion of the ninth, tenth, and eleventh nerves which is not fatal because of hemorrhage. The close relationship of the hypoglossal and sympathetic nerves to the ninth, tenth, and eleventh cranial nerves in the retroparotid space makes possible a very complex symptomatology.

The brief recital of the symptoms in one of Pollock's cases[1] will illustrate these lesions, which occur not infrequently in civil life. Like brachial plexus injuries, the original rather extensive paralysis may improve gradually until a permanent paralysis of one nerve with only a partial lesion of one or more of the others may be present as a residual lesion.

A year following a bullet wound, which coursed from just above the upper central incisors to a point 1 inch below and 2 inches behind the right mastoid process, a young man presented an enophthalmos, diminution of the palpebral fissure and a contracted pupil on the right side. In addition, he was unable to swallow solid foods, and there was a slight deviation of the posterior wall of the pharynx to the left. There was a history of regurgitation of fluids and hoarseness but no disturbance of respiration, pulse, or salivation. The right trapezius muscle was partially atrophied; the sternomastoid muscle was normal and the right half of the tongue was atrophied. The tongue deviated to the right upon protrusion.

It is obvious that in this patient, there was a permanent paralysis of the right hypoglossal nerve and the cervical sympathetic trunk, but in addition there was a dissociated and recovering lesion of the ninth, tenth, and eleventh cranial nerves. (Fig. 120.)

The external branch of the spinal accessory nerve, which innervates the trapezius and sterno-cleido-mastoid muscles may be injured alone. As a result, the sterno-cleido-mastoid muscle does not contract when the head is turned to the opposite side, and therefore, the head may be only incompletely turned. A complete division of the spinal accessory may be followed by only a partial paralysis of the trapezius muscle because it receives additional innervation from the cervical nerves. The usual deformity observed in these isolated lesions of the eleventh nerve is one in which the acromion falls, the upper and inner angle of the scapula is raised and displaced outward while the inner and lower angle is approximated to the midline. If the lower portion of the trapezius is paralyzed, the internal margin of the scapula is displaced outward, thus broadening the back, and the acromial portion of the clavicle is projected forward.

[1] These are reported in detail in Peripheral Nerve Injuries, Pollock, L. J., and Davis, Loyal, New York, Paul B. Hoeber, Inc., Chap. 36, 1933.

CHAPTER VII

SPINAL CORD INJURIES

"And truly they are more apt to lose the power of their legs and arms, to have torpor of the body, and retention of urine, who experience no displacement either forwards or backwards, but merely a violent concussion along the spine, while those who have displacement backwards are least subject to these symptoms."

HIPPOCRATES.

INJURIES to the back, particularly those which produce fractures or dislocations of the vertebræ, are complicated frequently by evidences of involvement of the spinal cord. In some of these cases the judgment of the physician is tested to its utmost to determine the proper pathological diagnosis and the best procedure to follow in treatment of the individual patient. The question is not only that of the indications for, the indications against, and the technique of laminectomy. Rather, it involves a problem of diagnosis and treatment wherein anatomy, neurophysiology, and neuropathology play a greater part than is at first apparent. Finally, these patients must receive medical, physical and vocational-industrial rehabilitation. Medical rehabilitation includes those aspects of care concerned with neurology, neurosurgery, urology, reconstructive surgery, nutrition, bone and joint surgery and psychology. The goal of physical rehabilitation should be the education and utilization of the patient's remaining muscle power. Vocational-industrial rehabilitation aims at integration of the patient as a useful member of society

With the exception of bullet wounds and rarely direct trauma to the back, nearly all of the injuries of the spinal column which are associated with damage to the spinal cord are the result of indirect violence which produces flexion or extension of the spine. As Stookey[1] has pointed out, there are two important factors involved: first, the architecture of the vertebræ, their mobility and that of the supporting ribs; and second, the blow, its force, direction, duration and point of impact.

The compact dorsal arch and the articulating processes are more resistant than the cancellous, relatively soft vertebral body, and therefore, the latter bears the brunt of the injury. Particularly is this true since in the majority of instances the indirect violence produces flexion of the trunk with transmission of the greatest force

[1] Stookey, B The Management of Fracture-Dislocations of the Vertebræ Associated With Spinal Cord Injuries, Surg., Gynec and Obst., 64, 407, 1937

ventrally. The force may be applied to the lower end of the spinal column, as is illustrated commonly in falls from a considerable height or in automobile accidents; or to the head, as is illustrated by diving into shallow water.

In flexion injuries, a compression fracture is the most common type produced. Part of the compressed vertebral body may protrude into the vertebral canal, or displacement of the body may occur with fracture of the articulating processes, usually the tips, and tearing of the articular ligaments. Comminution of the margins of one or both adjacent vertebral bodies may also occur with dislocation, and this is particularly dangerous to the spinal cord if the fragmentation and separation occurs along the dorsal or lateral borders. When fracture of the body and articulating processes occurs, the intervertebral disc is usually damaged, and this is followed by atrophy along the ventral margin with later angulation of the vertebræ.

From a consideration of the anatomical relationships between the spinal cord and the vertebral column, it is not difficult to understand the mechanism by which the spinal cord is damaged in fractures of the vertebræ. Nor is it difficult to understand how severe blows or jars to the spine may cause damage to the spinal cord with hemorrhages into its substance. In fact, the wonder is that of all the patients who receive fractures and fracture-dislocations of the vertebræ there are not more instances of injury to the spinal cord among them.

Embryologically the vertebræ and the intervertebral discs are developed about the notochord from mesenchymal condensations. In the adult the nucleus pulposus, a semiliquid substance, remains within the center of the intervertebral disc as a remnant of the embryonal notochord. The particular formation of the vertebral column gives it a certain flexibility, consisting as it does normally of 33 vertebræ, a few of them fixed, the majority more or less movable. The 5 sacral and 4 coccygeal units are fused to form 2 bones, whereas, the 7 cervical, 12 thoracic, and 5 lumbar vertebræ usually maintain their independence

The typical vertebra consists of an anteriorly placed oval body, flattened above and below, having a thin cortex surrounding a mass of spongy bone. Posteriorly the vertebral arch is made up of the pedicles and laminæ with a spinous process extending from it in the midline and paired transverse processes extending laterally. Various facets and processes serve to articulate the vertebræ with one another and with the ribs and thus permit at the same time a limited degree of flexibility of the column Certain variations from this typical pattern are of diagnostic and clinical importance. The extremely prominent spinous process of the seventh cervical vertebra makes it useful as a convenient landmark. The absence of a spinous process on the first cervical makes it impossible to palpate normally. In some individuals that of the second can be felt upon deep pressure. The spines of the third, fourth, and fifth cervicals are covered by fascia and muscles and are not felt so easily or distinctly. Through the mouth the bodies of the cervical vertebræ can be

felt as far down as the fourth and sometimes the fifth. In examining a fractured cervical region, however, this palpation should be done with extreme care because manipulation and examination may cause further displacement of the vertebræ and result in serious damage to the cord.

The spinal cord extends from the inferior margin of the foramen magnum to the lower margin of the first lumbar vertebra. The cord tapers conically to end in a slender filament (filum terminale) which extends to the end of the spinal canal. The conus medullaris may extend only to the twelfth thoracic or may be as low as the middle of the body of the second lumbar. In the embryo the spinal cord occupies the entire length of the canal. After the third

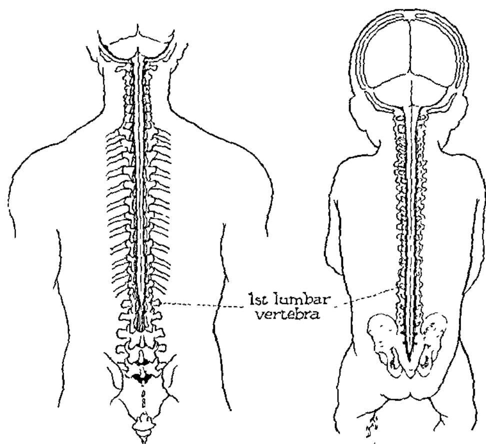


FIG 121.—Diagrammatic sketch showing the extent of the spinal cord in relation to the spinal canal in man and infant

month the spinal column grows more rapidly than the cord, and at birth the tip of the conus medullaris lies at the level of the third lumbar vertebra The final adult relationship is then attained more slowly.

From the cord thirty-one pairs of nerves arise. By passing an imaginary plane through the highest root filament of each spinal nerve the spinal cord may be divided arbitrarily into corresponding segments Accordingly, there are 8 segments in the cervical portion, 12 in the thoracic, 5 in the lumbar, 5 in the sacral, while there is only 1 coccygeal segment.

Each spinal nerve is formed by the union of an anterior or motor root, and by a posterior or sensory root which arise from each spinal cord segment.

These penetrate the dura separately with a thin septum of dura mater (ligamentum dentatum) between them, and unite outside the dura. In the cervical and upper thoracic regions the posterior root passes outside the dural sac at a right angle to the spinal cord. In the midthoracic area each root has a downward course until it reaches the dura mater. This downward and lateral course is more pronounced in the lower thoracic and lumbar portions of the canal. (Fig. 121.)

These anatomical facts are explained upon the discrepancy between the length of the spinal cord and that of the spinal canal. The lower the nerve root the greater the distance between its origin in the segment of the cord and its point of exit from the spinal canal. The lumbar and sacral nerves descend in parallel bundles to emerge 4 or 5 vertebræ lower than their level of origin, and it is these nerve roots which form the cauda equina. The bundles of nerves in the cauda equina are so arranged that the outermost roots are those which arise from the uppermost segments.

This relationship between the segments of the spinal cord and the vertebral bodies and spine is of surgical importance. In a general way the level of origin of the eighth cervical nerves lies beneath the sixth cervical spinous process. The twelfth thoracic segment of the spinal cord lies opposite the ninth spinous process. The fifth lumbar segment corresponds to the twelfth thoracic spinous process. In other words, there is roughly a difference of one between spinous processes and segments in the cervical region, two in the upper thoracic and three in the lower thoracic and lumbar regions. The sacral segments lie opposite the twelfth thoracic and first lumbar spinous processes. (Fig. 122.)

The degree of injury to the spinal cord is dependent upon two factors; the amount of displacement antero-posteriorly and laterally, and the rotation of the vertebra. In certain cases the roentgen-ray may show an apparently severe deformity which has produced spinal cord injury, but careful study will show more rotation of the vertebra on the long axis of the spinal canal than lateral or antero-posterior displacement. The laminæ, or the processes of the vertebræ, may be included in the fracture, but the future of the case depends upon the amount of injury to the cord since the fractures as such will heal as well as any other fracture of bone.

True anatomical damage to the cord may vary in degree from that caused by one or more small discrete areas of hemorrhage to larger lesions from gross hemorrhages. Other foci of damage may be more mechanical in type and vary from partial compression to complete transection of the cord in certain fracture-dislocations. While hematomyelia does occur in cases of injury to the spine without definite evidence of vertebral fracture, it is frequently possible to demonstrate some degree of traumatic bone damage by means of the roentgen-ray. This may be slight, such as a small chipping of the vertebral body near the level of the cord injury.

Cases of epidural and subdural hemorrhage with cord pressure in most traumatic cases cannot be clinically differentiated from other types of cord pathology. Especially is this true when, as is fre-

quently the case, these conditions are complicated by other injury to the cord. At times a slowly progressive extension of the initial paralysis and a gradual increase in the neurological findings may give a clue to such a situation. The presence of bloody spinal fluid,

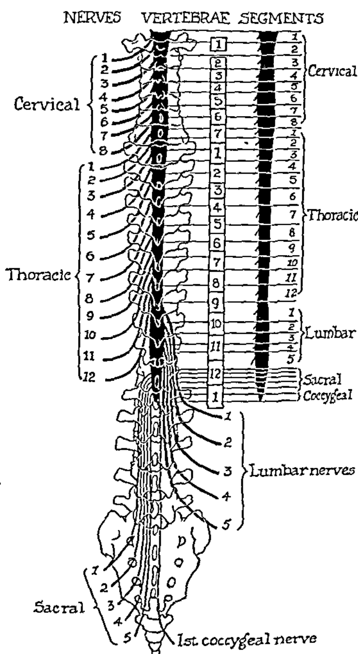


FIG 122. — Diagram which shows the relation of the spinal nerves and segments of the spinal cord to the vertebral spinous processes.

not a result of the lumbar puncture itself, is an indication of bleeding into the subarachnoid space, but considered alone it is not of sufficient evidence to warrant a diagnosis of a localized subdural hematoma.

Microscopically these cords may show as diversified a picture as the patients show clinically. At the site of the injury there is an initial edema and hemorrhage, varying from petechial to gross, within the substance of the cord. Early one can find swelling of the myelin sheaths, breaking up of the myelin, and later fragmentation of the axons. Phagocytes and scavenger cells of various origins, both from the blood (leucocytes) and from the nerve tissue (microglia, astrocytes) are in evidence, especially near and in the area of major damage. The ganglion cells undergo a change in cases of severe spinal shock even though recovery may occur. In severe local lesions the ganglion cells at the site of injury are involved to a greater degree; but even those cells some distance below the level of the lesion early present a picture not unlike that of beginning Wallerian degeneration, a state known as an axonal reaction. These cells usually recover and later may show no microscopic signs of previous damage.

Repair of damaged tissue in the central nervous system is by the formation of a glial scar made up mostly of astrocytes in its final state. Extensive scars at times may cause interference of function of neighboring undamaged tracts of the cord.

It goes almost without saying that every case of injury to the spine should be subjected to a careful and painstaking neurological examination. Only in this way is it possible to rule out damage to the nervous system. For this purpose a knowledge of some fundamental facts of neuroanatomy and neurophysiology and familiarity with a few important fiber tracts[1] is necessary for the simplest understanding of spinal cord lesions and the resulting symptoms. In addition, however, there are certain facts which must be kept in mind about the vertical localization of motor, sensory, and reflex functions in the different segments of the spinal cord.

If only a few of the motor segmental levels are kept in mind, they will serve as an aid in determining the location of a given lesion. For example, the motor segmental innervation of the deltoid muscles, which abduct and externally rotate the arm, is in the fifth cervical segment of the spinal cord. The biceps muscle is innervated by the sixth cervical segment, and the triceps by the seventh cervical segment. Therefore, given a patient with a spinal cord lesion who can flex but is unable to extend his forearms, one may state definitely that the lesion does not extend higher than the seventh cervical segment. Likewise, the abdominal recti muscles above the umbilicus are innervated by the ninth and tenth thoracic segments while the same muscles below the umbilicus are innervated by the eleventh and twelfth thoracic segments. Consequently, when the patient is asked to flex his neck with the hands by the side, if the umbilicus moves upward evidence is present of paresis or paralysis of the lower halves of the recti muscles. This is known as Beevor's sign and aids in determining the level of a lesion at the tenth thoracic segment.

[1] These facts were discussed in Chapter I.

The following table lists a few of the important motor segmental levels:

Muscle	Action	Level
Deltoid	Abducts and externally rotates arm	C–5
Biceps	Flexes forearm	C–6
Triceps	Extends forearm	C–7
Adductor longus Gracilis Adductor magnus	Adduct thighs	L 2–4
Tibialis anticus	Dorsiflexes foot	L–4

Closely associated with these motor segmental levels are the centers for the various reflexes, both deep and superficial, given in Chapter I.

Many anatomical, physiological, and experimental investigations have shown that the skin of the body is divided into zones in relation to the various segments of the body. If one imagines the body in the all-fours position of our ancestors and then makes transverse sections of the body in regular intervals beginning at the neck and passing to the coccyx, the segmental sensory distribution of the spinal cord will be understood more clearly. Because in this posture the thumbs and great toes are naturally in an advanced position, it will be readily seen that the corresponding sides of the arms and legs will be sectioned at a higher level than will be the opposite sides. For this reason the radial sides of the upper extremities are represented by a higher segmental level of the spinal cord than are the ulnar aspects. In the same manner the medial aspects of the thighs and legs are of a higher segmental level than are the external sides of the lower extremities. The successive segmental levels in the thoracic region are understood easily, and as one passes caudally the segmental representation of the primitive tail is about the anus. It is apparent at once that a loss of sensation which corresponds to a segmental area is diagnostic not only of a spinal cord lesion but of its level as well.

To these general diagnostic aids may be added the important symptoms characteristic of *upper* and *lower motor neuron lesions* referred to in Chapter I *Lower motor neuron lesions* occur as the result of injury or disease of the anterior horn cells, the anterior roots or the peripheral nerves, and only of those structures. They are accompanied by a flaccid paralysis, loss of deep tendon reflexes, muscle atrophy, and the reaction of degeneration as determined by electrical stimulation of the muscles. Of these three possible sites of a lower motor neuron lesion, the presence of sensory changes is diagnostic of a peripheral nerve lesion. An *upper motor neuron lesion* is characterized by the presence of a spastic paralysis, exaggerated deep tendon reflexes, absence of muscle atrophy, pathological reflexes (Babinski, Oppenheim, Gordon, Chaddock) and absence of

superficial reflexes (abdominal and cremaster). Such a lesion may occur at any level in the corticospinal tract. The occurrence of cranial nerve paralysis, involvements of other functional systems and the distribution of the motor weakness will determine the level of the lesion. For example, a paraplegia occurs rarely in cortical lesions but is common in spinal cord lesions.

SYMPTOMS

> "The prognosis during the first two weeks in any one case is extremely difficult, and it must be admitted that there is no one sign or symptom from which we can draw reliable conclusions on the severity of the lesion, or from which we can say, when there is complete motor and sensory paralysis, as there nearly always is in the earliest stages, whether the cord is completely divided or not."
>
> GORDON HOLMES.

It is unfortunate that there are no known criteria by means of which the doctor can differentiate clinically between a complete physiological and a complete anatomical interruption of spinal cord function. The former may be a more or less rapidly recoverable lesion, but the latter is an irreparable damage from which recovery does not occur. As in the case with peripheral nerve lesions during the early post-traumatic period a *complete physiological* interruption of the spinal cord cannot be differentiated from a *complete anatomical* section. Both are followed by complete paralysis in the muscles supplied by nerves which originate below the level of the injury. In both, complete sensory loss results below the level of the injured segment and the reflex changes and bladder disturbances may be similar. It is mandatory, therefore, that all cases of injury to the spinal cord, even those with retention of urine, paralysis of the extremities and loss of deep reflexes, be regarded and treated as though they were suffering merely from a temporary physiological block of function, rather than an anatomical section of the cord, until proved otherwise.

The neurological signs present after spinal cord injury may be due to edema of the cord, hemorrhage into the cord, compression of the cord by displaced vertebræ and anatomical section of the cord. Edema of the spinal cord occurs with every serious injury of the spine. It develops rapidly and is often marked. Within an hour after injury the spinal cord may be two or three times its normal size so that it fills the dural envelope tightly. This is the most common cause of manometric block (positive Queckenstedt test) during the first few days after injury. A manometric block, therefore, should not be considered as an indication for laminectomy during the immediate acute post-traumatic period. The clinical signs

may have observed only the terminal stage may have failed to observe any automatic reflex activity.

Certain differences of reaction in incomplete lesions may be pointed out; in complete lesions the flexor type of movements is observed commonly, while in incomplete lesions extensor types of movements are present frequently. In complete lesions, the posture of the lower limbs is one of slight flexion; in partial lesions, extension. As a general rule, partial lesions of the spinal cord show a condition comparable with that of a decerebrate animal in which there are defense reflexes with marked spasticity. Although an extensor type of response to plantar stimulation has been observed in complete section of the spinal cord, usually such a stimulation is followed by a plantar flexion of the toes, and as a fairly general rule it may be stated that an extensor type of reflex is strongly indicative of an incomplete lesion. Inasmuch as prolonged states of toxemia or septicemia from urinary sepsis or bed sores have a profound influence in hastening the reappearance of the reflex inactivity in cases of complete section of the spinal cord, it frequently occurs that from the practical standpoint incomplete lesions are relatively easily recognized by the long persistence of spasticity and the signs of a paraplegia in extension.

Of particular value in the recognition of incomplete lesions is the early appearance of a Babinski sign; the failure to evoke mass reflexes from above the knee; a definite history of the absence of a state of spinal shock; marked tonicity in the paralyzed extremities; the involvement of both flexors and extensors in reflex movements provoked by the stimulation of receptive fields; and, of course, in obviously incomplete lesions, the absence of total paralysis or anesthesia below the level of the lesion.

We have had an unusual opportunity to study carefully and under a good plan of organization, 793 patients with spinal cord injuries received, in the majority of instances, in World War II. Due to the high quality of their medical care, many of these patients are alive 10 years after their injury was received, a circumstance not uncommon, of course, in patients injured in civilian life.

In the cervical spinal column a forward dislocation of the head and the upper portion of the cervical spine over the lower portion usually occurs. This is accompanied by a fracture of one or more of the articulating processes of the affected vertebræ. The essential lesion, however, is a forward dislocation.

Although we have not observed such a case as yet, injuries of the cord opposite the first and second cervical vertebræ are almost invariably and immediately fatal. However, patients with such an injury may remain alive for a few hours or days with marked bulbar symp-

toms and with pain in the distribution of the upper cervical nerves. Death may occur suddenly as a result of a movement of the head as in sneezing or coughing. Fractures of the third and fourth cervical vertebræ cause death in most instances from respiratory paralysis or the development of pneumonia, and this has been our experience in spite of the early use of oxygen therapy.

Injuries in the region opposite the cervical enlargement of the cord show characteristic symptoms. In all of our cases there was a more or less complete paralysis of the muscles of the lower extremities and the trunk with a partial paralysis of the muscles of one or both upper extremities. If the fifth and sixth cord segments are not injured, the deltoid, biceps, brachialis anticus and supinator muscles are not involved although the muscles of the forearms and hands may be paralyzed. As Thorburn pointed out, these patients lie with the arms abducted, the forearms flexed and rotated outward. The power of the upper extremities may be preserved in injuries below the first dorsal vertebra although weakness in the triceps can usually be detected. In one of our patients injured in an automobile accident, a fracture-dislocation of the fifth cervical vertebra had produced an immediate and complete loss of motion and sensation in both arms and legs. Within three weeks a slight degree of motion had returned in his left arm and leg, and on this side below the clavicle there was a complete loss to pain and temperature stimuli. Sensation on the right side was intact though motion was completely absent. This interesting group of symptoms, first described by Brown-Séquard, is indicative of a unilateral lesion of the spinal cord.[1] (Fig. 123.)

Severe pain in one or the other shoulder is prominent in injuries of the sixth cervical to the first dorsal vertebræ, and there may be root hyperesthesias or anesthesias over the shoulder, in the axilla, or on the radial or ulnar sides of the upper extremities. If the lesion extends up to the fifth or sixth cervical segment, the sensory disturbances will affect the radial side of one or both forearms; but if it does not extend above the seventh cervical segment, only the ulnar sides of the upper extremities are involved. Priapism is frequent in injuries of the cervical cord, and an elevation of the temperature to 107° or 108° F. is quite characteristic. This type of hyperthermia is not affected by the common therapeutic measures employed to lower the temperature in other conditions.

Lesions of the brachial plexus may often accompany compression injuries of the cervical spinal cord. These are usually of two clinical

[1] Brown-Séquard's syndrome (motor paralysis upon the side of the lesion and loss of temperature and pain sensation *on* the opposite side) is more commonly produced by spinal cord tumors, stab or gunshot wounds of the cord.

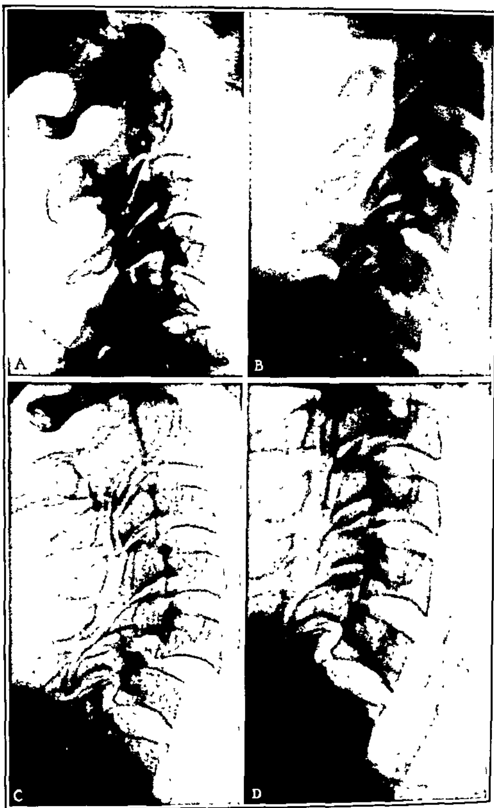


Fig 123 —(*A*) Roentgen-ray film of the cervical spine which shows posterior dislocation of the fifth cervical vertebra, (*B*) roentgen-ray film of the cervical spine which shows fracture dislocation of the sixth and seventh cervical vertebræ, (*C*) posterior dislocation of the sixth and seventh cervical vertebræ with slight comminuted fracture of the anterior surface of the body of the seventh vertebra, (*D*) reduction of the dislocation in the same patient forty-eight hours following traction by skull tongs

types. There may be an immediate paralysis of all four extremities, which is quickly followed by the return of motion in one arm and leg. The opposite arm then presents a flaccid paralysis, the corresponding leg a spastic paralysis, and there is loss of pain and temperature sensation on that side of the body in which motion has returned to the extremities. The spastic paralysis of the leg may eventually disappear, and a residual flaccid paralysis of the upper extremity remains.

In another clinical type, the patient may sustain an injury to the brachial plexus and then several days or weeks later may develop a slowly progressive paralysis of the leg on the same side, with loss of pain and temperature sensation on the opposite side. In these cases it is necessary to determine whether the flaccid paralysis of the arm, distinctly a lower motor neuron lesion, is due to an injury to the nerves of the plexus, to the roots, or to the anterior horn cells of the spinal cord.

If the lesion is one of the plexus itself, the loss of touch sensation will be greater than the loss of pain. If it affects the roots, the loss of pain sensation will be more extensive than the loss to touch. If only the anterior horn cells are involved, sensation will be unaffected. However, if sensory loss exists with such a lesion of the gray matter, then light and pressure touch as well as pin prick and pressure pain will be absent. Finally, if the paralysis in the leg disappears relatively soon and the arm paralysis persists, it speaks for a brachial plexus lesion as against one of the spinal cord.

In about 48 per cent of cervical cord injuries spastic paraplegia in flexion occurs and 13 per cent have flaccid extension. Seventy per cent of all cervical injuries have spasticity but 98 per cent have pseudospontaneous spasms. The pattern of the latter differs from responses evoked by exteroceptive stimuli in that there is a preponderance of bilateral simultaneous similar movements either in flexion or extension. The extensor movements occur twice as frequently and adduction of the thighs three times as frequently as abduction, whereas in induced reflexes, they are equal.

In a severe transverse lesion of the cervical spinal cord, suprasegmental impulses are interrupted on their way to the thoracicolumbar sympathetic outflow and to skeletal muscle. Aside from any possible hormonal effects, no central excitation for heat production is effective. Likewise no central inhibition of heat production is effective; neither are central thermo-regulating impulses affecting heat loss by sweating, or shift of blood volume, effective. Such regulatory functions as are produced in these patients reside in the distal segment of the spinal cord and are subject only to local reflexes.

Thus, having lost the regulatory function such an individual becomes partly poikilothermic.[1]

Central excitatory, inhibitory and regulatory impulses for water metabolism do not reach the appropriate levels when the cervical spinal cord is severely injured. Whatever effective neural function remains of water metabolism resides in the distal segment of the injured spinal cord, with the possible exception of an hormonal effect. The increased temperature of the surrounding air in hot weather serves as an adequate stimulus in these cases for an increase of urinary output in excess of the normal.

In cervical cord lesions, sweating does not result from stimuli which act centrally, as does heat, since impulses from levels above the origin of the thoracico-lumbar sympathetic outflow are interrupted. However sensory impulses from distention of the urinary bladder reach the thoracico-lumbar sympathetic outflow and sweating occurs in some parts over the body.

The regulatory vasoconstrictor, vasotonic and vasodilator impulses from the cortex, diencephalon and medulla are interrupted in their pathway to the thoracico-lumbar and sacral sympathetic outflow in the presence of cervical spinal cord lesions. Consequently, changes in blood pressure which result from reflex activity in the distal segment of the injured spinal cord are uninhibited and they assume great proportion and last a longer time. Sensory impulses from the application of cold to the hand do not produce a significant rise in blood pressure in these patients but, on the other hand, stimulation of the feet by cold produces a sustained increase of blood pressure in both the arms and legs.

In cervical cord lesions, sensory impulses which result from distention of the bladder and rectum are able to reach the thoracico-lumbar sympathetic outflow and a rise in blood pressure results. Because of a loss of regulatory influence of upper levels, tilting from the horizontal position through varying degrees to the vertical, results in such a marked drop in blood pressure that it may be impossible to measure and eventuates in loss of consciousness. Since the glossopharyngeal-vagus regulatory function is undisturbed, the relationships of increase of heart rate and lowering of blood pressure

[1] The extensive and meticulous studies made by Dr. Lewis J. Pollock and his colleagues on the spinal cord injury patients at the Veterans Administration Hospital under the medical supervision of Northwestern University have added a tremendous amount of important, new knowledge about spinal cord injuries

Pollock, Lewis J., *et al*: Pain Below the Level of Injury of Spinal Cord, Arch. of Neur. and Psych., 65, 319, 1951.

Pollock, Lewis J., *et al*: Management of Residuals of Injury to the Spinal Cord and Cauda Equina, Jour. A.M.A., 146, 1551, 1951.

Pollock, Lewis J., *et al*: Defects in Regulatory Mechanisms of Autonomic Function in Injury to Spinal Cord, J. Neurophysiol., 14, 85, 1951.

and slowing of heart rate with increase of blood pressure remain unchanged.

Thoracic Cord Injury.—In the thoracic spine, the chest cage affords a splint so great that fractures severe enough to produce signs of spinal cord injury rarely occur unless the trauma has been extremely violent.

The rather characteristic symptoms produced by involvement of the twelfth thoracic vertebra are illustrated in the following case as are the dangerous results which are likely to follow unskillful attempts to reduce or "adjust" such dislocations. (Fig. 124.)

A young woman fell down a flight of stair steps and immediately lost control of her legs and sphincters as well as suffering a loss of sensation to all stimuli below the level of the tenth thoracic skin segment. At the time of her injury she was four and a half months pregnant but successfully delivered her baby at term. For two years she was subjected to attempts designed to reduce a dislocation of the twelfth thoracic vertebra. When she entered the hospital, the motor and sensory findings were unchanged, the deep tendon reflexes of the lower extremities were increased, pathological reflexes were present, and the lower abdominal reflexes were absent.

A complete spinal subarachnoid space block was present, and at operation the spinal cord was found to be compressed by the fractured twelfth thoracic vertebra. The dura mater was thick; the arachnoid was thickened and inseparable from the underlying traumatized spinal cord.

Loss of the abdominal reflexes and a loss of sensation over the abdominal wall corresponding to the sensory distribution of the segments involved are characteristic of lesions in the lower half of the thoracic spinal cord. Since the upper halves of the abdominal recti muscles are innervated by the eighth to tenth and the lower halves by the tenth to twelfth segments, Beevor's sign may be present. If the patient raises his head from the bed with his arms at his side, the umbilicus is pulled upward if the lesion is at the level of the tenth segment because of the paralysis of the lower halves of the recti. Likewise, the upper abdominal reflexes on one or both sides may be preserved while the lower are absent. Girdle-like root pains are most frequent in injuries of the mid-thoracic vertebræ.

Paraplegia in flexion occurs in about 35 per cent of the patients with injuries of the spinal cord from the first to the ninth thoracic segment inclusive and about 13 per cent show a flaccid extension. Sixty per cent of these patients have spasticity and 95 per cent show pseudo-spontaneous spasms.

In thoracic cord lesions, sweating which results from centrally acting stimuli, such as heat, may occur in areas below the level of the injury to the spinal cord since preganglionic sympathetic fibers from above enter into a plexus formation with those below. Reflex sweating resulting from the sensory impulses of a distended bladder

may be found in areas above the level of the injury to the cord. This is due to plexus-like connections from lower portions of the sympathetic chain with upper ganglia and grey rami.

A lesion which produces a hemisection of the spinal cord (Brown-Sequard) results in paralysis of the thermoregulatory sweating below the level of the injury. Central sweat regulatory fibers pursue a



Fig 124 —Fracture dislocation of the eighth and ninth thoracic vertebræ sustained in an automobile accident The patient suffered a complete physiological lesion of the spinal cord.

unilateral course through the spinal cord on the side of the corticospinal tract injury Therefore, sweating is lost on the paralyzed side below the lesion.

Sensory impulses which result from cold stimuli applied to the foot cannot reach the thoracico-lumbar sympathetic outflow to the upper extremities, therefore, in injuries of the mid-thoracic cord there is no rise in blood pressure in the arm, but there is in the leg.

Cauda Equina Injury.—In the lumbar spine following sudden arrest of forward motion of the patient's body, the strong articulations between the vertebræ hold fast, but the cancellous bone of the vertebral bodies gives way. The resulting lesion is most frequently a compression and forward angulation.

Attention has been called to the fact that the spinal cord ends opposite the superior border of the second lumbar vertebræ and that below that level the spinal canal is occupied by the cauda equina. Consequently, the symptoms of a lower motor neuron lesion are frequently characteristic of an injury to the lumbar vertebræ. Flaccid paralysis, loss of the deep tendon reflexes, muscle atrophy, reaction of degeneration, and loss of sensation in the area of distribution of the lumbar and sacral segments are the usual symptoms. However, because of the nature of the cauda equina, involvement of motor and sensory function may be very asymmetrical in the lower extremities. In one of our patients who had a compression fracture and an anterior dislocation of the first lumbar vertebra, the most extensive sensory loss was in the second and third lumbar skin segments on the left side. Likewise, many of the muscles of the right leg continued to react to the faradic current; whereas, in the left leg faradic reactions were completely lost.

Retention of urine and incontinence of feces are prominent symptoms. Excruciating pains in the back or radiating pains into the perineum, genitals, and often down the posterior surfaces of the thighs occur frequently as the result of root irritation produced by the fragments of fractured bone. (Fig. 125.)

A study of the 793 patients in the Veterans Administration Hospital, in addition to those we have cared for in civilian practice, has led to certain conclusions concerning other symptoms which are commonly encountered regardless of the level of the lesion. It is necessary to understand completely each individual patient's symptoms and to be able to explain them to him satisfactorily if he is to make a successful rehabilitation.

The *pain* which follows spinal cord injury has been found to be of much higher incidence in military than in civilian patients, probably because of the preponderance of open injuries in the former. The pain which accompanies either partial or complete lesions of the spinal cord falls into three general categories: root pain, visceral pain, and a diffuse, poorly localized, burning or tingling pain. The mechanism of production of root pain, as well as the means of its elimination by proper surgical procedures, is well understood. The pathways for the transmission of the visceral types of pain along short neurons adjacent to the gray matter are likewise well understood, but these sensations do not require surgical intervention. The pain described

as *burning, tingling,* or *electric-like* is never well localized by the patient and it has never been found to follow the pattern of any peripheral nerve, or particular dermatome. Emotional and environmental factors play an important role in its mechanism.

Peripheral physio-chemical changes in the skin and muscles initiated by an irritative focus in the distal injured segment of the spinal cord may originate impulses which are spread upward through the



FIG. 125.—Fracture dislocation and compression of the second, third, and fourth lumbar vertebræ with marked angulation and displacement. The patient presented symptoms of a cauda equina lesion.

autonomic system to enter the cord over a wide distribution and eventually reach the thalamus and cortex to be recognized as diffuse, burning pain. Or, this pain may be due to direct stimulation of the pain fiber tracts within the proximal injured segment of the spinal cord.

It has been proposed that suitable stimulation of structures innervated by segments distal to the level of the lesion may result in pain because the impulses find their way into the proximal segment of the spinal cord by way of the sympathetic nerve fibers. Such sensations have been said to have been evoked from stimulation of subcutaneous structures, viscera and blood vessels. Even faradic stimulation of the skin has been reported to produce pain. The observations we have made in this large group of patients completely controverts the presence of such pathways for pain; in other words, stimuli designed to produce pain from blood vessels, bones, or other subcutaneous tissues and viscera, send impulses over the ordinary sensory pathways in the spinal cord.

Be the injury cervical or thoracic, open or closed, with a complete lesion of the spinal cord as indicated either by surgical verification or by the long persistent failure of recovery, the severity of the *heightened* reflexes varies from patient to patient. There are those who never at any time develop anything more than the mildest degree of spasm, while others early and rapidly progress to such a condition of hyperactive reflexes that the persistent deformity calls for specific medical or surgical care. It has been our observation that the early care of the patient has much to do with his later period of heightened reflex activity. When the proper surgical care has been given early, and when all supportive measures, such as early ambulation of the patient, physical therapy, proper care of the bladder, the avoidance of decubiti and the other sources of sepsis and general debilitation have been attained, our patients have been spared later excessive reflex activity and all the attendant ills.

It is commonly stated that *perspiration* does not occur below the level of a complete lesion, yet we have many times seen such profuse, dripping perspiration over the buttocks and thighs with lesions as high as the fourth thoracic segment as to be a threat to the development of macerated skin and of decubiti.

We have also observed, especially in upper thoracic lesions, that a full bladder, just before its automatic evacuation, or a bowel distended by an enema, may produce an extensive flush of the body above the lesion, headaches, perhaps nausea, "gooseflesh" on the skin of the lower extremities, and finally, on evacuation, a return to a normal color of the skin of the face, neck, and arms, with the sudden appearance of beads of sweat over the thighs and trunk. In many

patients these same visceral responses may follow strong stimuli which produce flexor activity, such as pricking of the soles of the feet.

We have found that after the first 8 or 10 days following injury, during which the bowels are usually constipated, manipulations of the lower extremities, bathing the patient, or changing his position, may result in the sudden evacuation of the bowels. As the months wear on, however, the bowels develop a tendency to either spontaneous evacuation or evacuation after enemas or digital stimulation of the rectum, but in any case with a decreasing tendency to take part in the generally heightened reflexes.

FIRST AID CARE

The laity has been educated very little concerning the methods which should be employed in giving first aid care to patients who may have an injury of the spinal column. Yet there is no other group of patients in whom careless handling or transportation may convert a relatively slight injury into an irremediable one more quickly. Stookey has insisted that the lay public must be constantly instructed to realize that anyone unable to use his arms and legs, or the legs alone, should not be moved until competent aid arrives.

The most important principle in the emergency treatment of fracture-dislocations of the spine is to do nothing which will increase the bony deformity. The patient should not be moved unless it is absolutely necessary because each new movement may cause bony fragments to cut into the spinal cord. The patient's head should not be raised to give him a drink or a cigarette. A rolled blanket or pillow should not be placed beneath the head. He should not be lifted off the ground unless he is on a litter or other rigid support. Reduction should not be attempted. Plaster casts should not be applied.

The patient with a fracture-dislocation of the cervical spine is in the optimum position when he is lying on his back, with a folded blanket 3 to 4 inches thick beneath his shoulders, with his head below the level of his shoulders and his neck in slight dorsiflexion (hyperextension). Folded blankets should be secured at both sides of the head to prevent lateral movement. He should not lie face down with his neck twisted, nor upon his side with his neck flexed laterally. Care should be taken that clothing and blankets are smooth beneath the patient. Pockets should be emptied.

Three persons are needed to turn properly a patient with a fracture-dislocation of the neck onto his back from some less favorable position. One should grasp the chin and occiput and exert steady trac-

tion in the line of the long axis of the body. The second should grasp the ankles and exert equal countertraction along the same line of pull. The third should then kneel beside the patient, reach across his body and grasp the patient's clothing near the shoulder and near the hip joint with his two hands. He should then gently rotate the patient toward himself while the men at the head and feet exert their traction. The head, neck, body and legs should be so placed on the ground or the litter that the patient's shoulders finally come to rest on top of it when rotation has been completed. This position will allow the head to hang down slightly and permit a moderate dorsiflexion (hyperextension) of the cervical spine, a position which tends to correct the deformity caused by the fracture-dislocation. If the patient is lying face upward on the ground, the three men should take the same positions as for turning him, with the exception that the man at the side should grasp the patient's clothing on the side nearest to himself. The three men then gently slide the patient from the ground on to the litter without lifting him. The two men at the head and feet should meanwhile maintain a strong longitudinal traction.

Morphine should not be given to patients with injuries to the cervical spinal column.

All patients with fracture-dislocation of the cervical spine should be transferred immediately to a hospital where definitive neurosurgical treatment can be given.

The fundamental principles for the emergency treatment of compression fractures of the lumbar spine are essentially the same as for fractures of the cervical spine; the patient should not be moved unless it is absolutely necessary. He should not be picked up and carried from one place to another unless he has first been placed upon a litter or other rigid structure. There are two acceptable positions for a patient with a compression fracture of the body or one of the lumbar vertebræ. If an air mattress is available, the patient should be placed upon it, lying upon his back, with two or three folded blankets underneath the air mattress at the site of the fracture so placed as to produce hyperextension of the spine. If an air mattress is not available then the best position is the face-down position. This position automatically prevents further anterior flexion of the lumbar spine. It also prevents the formation of pressure sores at the point of angulation and over the sacrum. No other attempts at correction of the bony deformity should be made.

The principles involved in turning a patient with a compression fracture of the lumbar spine are similar to those for turning a patient with a fractured cervical spine, except that in fractures of the lumbar spine, traction by the man at the head of the patient may be applied

under the armpits. In transferring the patient from the ground to the stretcher the same principles apply as in the case of a patient with a fracture-dislocation of the neck.

Compound injuries of the spinal column are usually the result of gunshot wounds. No attempt at definitive treatment should be made at the site of the accident. The antibiotics and sulfonamides should be used locally and systemically at once and intensively, particularly if the cerebrospinal spaces are exposed. Large sterile protective dressings should be securely applied. In all other respects the patient should be treated and transported, in accordance with the principles outlined for the care of closed spinal injuries, to a hospital where definitive neurosurgical care can be given and the soft tissue wounds cleansed thoroughly with soap and water, surgically excised and treated. Since penetrating wounds of the spine are frequently associated with wounds of the lungs, or abdominal viscera, these injuries should be examined for and treated appropriately.

Laminectomy with full exposure of the cord is usually indicated in the treatment of compounded injuries of the spinal column. Splinters of bone and metal should be removed from the spinal canal when their presence threatens damage to the spinal cord. The dura mater should be closed, if possible, to prevent spinal fluid leakage. A free transplant of fascia may be used to accomplish this if necessary. The muscles, fascia, subcutaneous tissue and skin should be carefully closed layer by layer without drainage, otherwise a spinal fluid leak with the formation of a fistula and a terminal meningitis is apt to occur.

SURGICAL INDICATIONS

There are two problems which must be solved in the decision for or against surgical intervention in fracture-dislocations of the spine. First, is there a demonstrable block of the subarachnoid space, which means compression of the spinal cord? Second, is there roentgen-ray evidence of bony encroachment upon the spinal canal? Positive evidence of subarachnoid block usually implies bony encroachment on the spinal canal. It must be emphasized that a complete anatomical lesion of the spinal cord cannot be differentiated from a complete physiological section. Therefore, unless one has inspected the cord at operation, there can be no justification for the assumption that the cord has been divided completely. Surgical intervention may be useless and often harmful in the presence of a complete transverse anatomical lesion of the spinal cord; but, as has been pointed out, a complete transverse physiological lesion may be present from compression by a dislocated vertebra. It would appear that the benefit which may be derived from operative interference

is based simply upon the question of whether or not there is pressure upon the cord by a dislocated vertebra or fractured fragments of bone. In the presence of such pressure there is no question as to the benefit to be gained by an operation. Perhaps the most valuable aid in addition to the roentgen-ray and a careful physical examination is the employment of the Queckenstedt test of pressure upon the jugular veins.

The dural sac, which encloses the spinal cord, is continuous with the dural covering of the brain. This dural sac ends at the lower border of the second sacral vertebra; below this level the dura mater closely invests the filum terminale and descends around it to the back of the coccyx. The spinal dura is separated from the arachnoid membrane of the cord by a potential cavity only. The spinal and intracranial subarachnoid spaces are thus normally continuous. Because of this fact, pressures within the cranial cavity are normally transmitted through the medium of the cerebrospinal fluid to the spinal subarachnoid space. This makes possible the verification of suspected spinal compression. The normal pressure of the cerebrospinal fluid at the lumbar region, in the lateral recumbent position, is from 10 to 18 cm. of water. In the sitting or upright position the pressure is higher, depending to some degree on the height of the column of fluid thus superimposed above the puncture site. Coughing causes a definite elevation in pressure, as does straining or strong pressure upon the abdomen.

Compression of the jugular veins in the neck of a normal individual causes a definite prompt rise in the intracranial fluid pressure (Queckenstedt's sign). This rise in pressure is transmitted normally to the fluid in the spinal subarachnoid space. Release of jugular compression in normal subjects is followed by a prompt return of the pressure to approximately its original level. When, however, this increase in pressure does not take place on compression of the jugular veins, it is indicative of an obliteration of the spinal subarachnoid space.

Cases of partial block may be recognized by the slowness with which the pressure changes in the spinal system after compression of the jugular veins, the less than normal rise produced, and the comparatively long time it takes for the pressure to return after release of the jugulars. The efficiency of the needle and manometer system can be checked by having the patient cough or strain. This procedure will raise the pressure in the spinal subarachnoid space independently though a block is present.

Normally the spinal fluid is a clear, colorless liquid. In cases of long-standing obstruction, the fluid below the block becomes canary yellow in color. On standing, it coagulates into a jelly-like clot, and there is a marked increase in the protein content. This is known as Froin's syndrome. In more recent obstructions the total protein content of the spinal fluid, which normally varies between 25 and 35 mg. per 100 cc., will be elevated.

Often the character of the dislocation or rotation of the fractured vertebra as shown upon the roentgen-ray films is in itself sufficient

indication for surgical intervention. However, occasionally symptoms of cord injury may be present in the absence of roentgen-ray evidence of bone pathology. In such instances we have come to depend entirely upon the presence or absence of a subarachnoid space block, after spinal cord edema has subsided and in each of our patients operated upon a manometric block has been demonstrated. It is justifiable to perform successive Queckenstedt tests to follow the persistence or disappearance of the block. This is true particularly if there are clinical evidences of improvement in the symptoms. Likewise, in the presence of demonstrable roentgen-ray evidence of bone pathology and in the absence of a subarachnoid space block, one is justified in waiting for a spontaneous improvement of symptoms. In other words, the demonstration of compression of the spinal cord is the indication for surgical treatment and not the supposed extent or character of the spinal cord damage.

Other diagnostic aids may be used to determine the level of the lesion and the type of pathology present. The use of radiopaque contrast substances like pantopacque may be of value in some selected cases with doubtful clinical findings and evidence of a partial block after an ancient injury. The injection of air into the spinal subarachnoid space as a contrast medium has been advocated for the localization of the level of a complete block. It has the advantage of not being so irritating as the heavy contrast oils, but it does not provide accurate evidence in cases of partial spinal fluid block.

With such preliminary understanding of the anatomical, neurological, and diagnostic methods and mechanisms involved in the cases of injuries to the spine with spinal cord damage, the indications for surgical intervention become less involved. We may say first of all that there is no immediate indication for surgery in the case of injury to the spine in which there are no signs or symptoms of the spinal cord involvement.

The mere fact that there are signs of neurological damage in injuries of the spine does not, however, constitute a surgical indication. In cases of spinal injury with minor neurological symptoms or signs of a recovering lesion and no evidence of subarachnoid space block, treatment by conservative measures is the method of choice.

With manifestations of neurological involvement of a definite degree where there is compression sufficient to cause a subarachnoid space block, there exists a definite indication for laminectomy. It is conceivable that certain cases may present themselves with major neurological signs and roentgen-ray evidence of severe bone injury (crushing and fragmentation with spicules driven into the spinal canal) which do not give evidence of a block on manometric study. These cases should certainly be operated upon.

There remain some patients who have a questionable amount of permanent neurological damage, questionable block, and questionable roentgen-ray evidence of encroachment upon the spinal canal. It is in these cases that perhaps the definite judgment of the neurologist and surgeon is tested to its utmost. With these patients it is probably better to err on the side of a possible unnecessary operation.

With the knowledge that the spinal cord does not regenerate once it is anatomically severed, even if one believes in certain cases that the displacement and fragmentation of the bone has been so great as to most certainly transect the cord, these patients should not be denied an opportunity for recovery. In some, recovery will occur when the pressure is relieved, and no one can tell by clinical examination alone that a complete irreparable lesion is present.

Occasionally a patient presents himself with evidences of spinal cord damage at a time after the injury to the spine when the bony damage has completely healed. The question of surgical intervention in these cases is at times a delicate one. Here so much depends upon the nature of the lesion, the degree of disability it is causing, and the course of its progress that no inclusive rules can properly be laid down. In these cases every effort should be made to arrive at an accurate pathological diagnosis before submitting the patient to an operation. Then probably only those cases with lesions which offer some chance for surgical relief should be operated upon. At times we have been forced to a decision as to whether or not to explore an apparently hopeless lesion.

Undoubtedly, there is a very definite relationship between the level of the injury and the mortality rate. The higher the injury in spinal level the higher the mortality rate. Likewise, the more severe the degree of neurological damage, the higher the mortality rate. This latter is especially true in lesions of the upper dorsal and cervical region.

The time of operation after receipt of the injury is important and is dependent upon the demonstration of a subarachnoid space block in addition to the physical condition of the patient. One may with justification wait for several hours until the immediate state of general shock has subsided before a laminectomy is performed; but this period should not be extended to the point of waiting for the cessation of symptoms of improvement in the presence of a block. Though considerable recovery may occur spontaneously, the mortality of the operation of laminectomy in competent hands is too low to justify such conservatism.

Fracture-dislocation of the cervical spine, with or without a subarachnoid space block, is best corrected by closed traction methods, until it has been shown that the subarachnoid space block has not

been relieved. Laminectomy, early after trauma, is contra-indicated because of the high mortality rate.

The best method of reduction is by skeletal traction applied with the skull tongs described by Crutchfield. Halter traction with straps applied around the occiput and beneath the chin is unsatisfactory because it is extremely uncomfortable, it interferes with movements of the jaw in eating and talking, and it tends to produce pressure sores beneath the chin and at the occiput. Furthermore, considerably less traction may be applied by this method. The former practice of rapid reduction, using traction of considerable force, with the immediate application of a plaster cast extending over the occiput and chin down over the thorax, is no longer justifiable with the newer methods now available. Frequent *x*-ray examination should be used to control the reduction of the fracture-dislocation at all stages, both early and late. Traction should be maintained until sufficient time has elapsed for firm fibrous union to be established between the injured vertebræ, and this usually requires at least six weeks, and in severe injuries twelve weeks. Weight bearing should be deferred until repair of bone is well under way. Manometric studies upon the spinal fluid should be performed as often as necessary to check the effectiveness of the treatment.

Fracture-dislocation of the thoracic spine with gross misalignment of the vertebræ cannot be satisfactorily corrected by any method, open or closed. Pressure upon the spinal cord can be removed surgically by a laminectomy.

Compression fractures of the lumbar spine with angulation and deformity of the spinal canal often do not require surgical procedures. Frequently, these deformities are best corrected by closed methods of hyperextension but again manometric spinal fluid studies are extremely important. Fracture boards should be placed upon the bed to present sagging. Two or three blankets should be made into a roll the width of the bed and approximately 18 inches in diameter. This is placed across the bed on the fracture boards at the level of the spinal injury. A hard, hair mattress is placed over the blanket roll and on top of this a softer mattress, preferably an air mattress. The curved surface of the uppermost mattress will then conform approximately to the normal lumbar curvature of the healthy spine. When the fracture bed has been thus prepared, the patient is gently lifted on to it, face up, in such a position that the site of the injury lies above the blanket roll. The weight of the upper and lower parts of the body will then serve slowly to bring the kyphosed spine into a position of normal lumbar lordosis. This method of reduction is far more efficient and less traumatizing, mentally and physically, to the patient than the older method of reduction by suspension upon a canvas hammock (Fig. 126.)

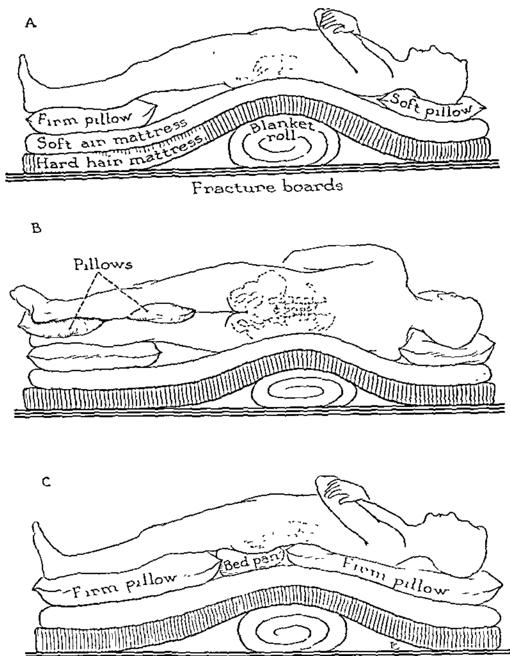


FIG 126.—Care of the patient with compression fracture of the lumbar spine. (*A*) Position of rest. (*B*) Position on the side. (*C*) Method of using bed pan. (Courtesy of Dr. John E. Scarff and Surg., Gynec. and Obst.)

NURSING CARE

The bed care of patients with a fractured spine is of the greatest importance In order to change bed linen or to bathe these patients, they may be turned on their side without risk of injury if the bed has been properly set up. The soft tissue between the hip, the pelvis and the lower edge of the thorax will conform very well to the curva-

ture of the bed as the patient is turned without permitting significant lateral misalignment of the spinal column. Moreover, the large intervertebral articulations at the level of the lesion are rarely damaged sufficiently to allow lateral misalignment.

In using the bed pan the patient is rotated gently to one side. The bed pan is then placed in the center of the bed and firm pillows or folded blankets having the same thickness as the bed pan itself, are placed on the bed above and below it. If these pillows or blankets have the correct thickness and firmness and are properly placed, the original curvature of the top mattress is preserved. The patient is then slowly rotated on to his back so that the back and legs are supported at exactly the same level as the bed pan. In this way hyperextension of the spine is maintained A bed pan should never be placed beneath a patient with a compression fracture of the lumbar vertebræ unless these precautions against antiflexion are first taken. Another method of evacuating the bowels of these patients is to place them upon one side and carry out colonic irrigation.

Prevention of hypostatic pneumonia is a major problem during the first days of treatment in all fractures of the spine. In fracture-dislocation of the cervical spine injury often occurs to the fourth and fifth cervical segments from which the phrenic nerves innervating the diaphragm take origin. In addition to this the edema of the cord tends to interfere with the passage of impulses from the respiratory center of the brain to the respiratory musculature of the thorax. For these two reasons respiratory movements are apt to be shallow. Coupled with this is the fact that the patient is lying on his back, in which position it is difficult for him to clear his mouth and posterior pharynx of accumulated mucus. Should signs of impending pneumonia appear, it is imperative that treatment of the pneumonia take precedence over treatment of the fracture. Accordingly these patients must be turned from one side to the other every two hours, day and night, to permit free drainage of exudate and mucus from first one-half and then the other half of the respiratory tree All other measures generally employed in the treatment of pneumonia should, of course, be utilized.

In compression fractures of the lumbar spine pneumonia is caused by a different mechanism. The continued hyperextension of the body causes the rectus muscles of the abdomen to pull upon the lower ribs. An acute traumatic periostitis frequently develops at the point of attachment of the muscles. This may be quite painful and causes involuntary splinting of the lower part of the thorax. This limitation of respiratory movements in the lower lobes of the lungs predisposes to the development of hypostatic pneumonia in

much the same way that splinting of muscles after upper abdominal operations does. These patients must be given sufficient sedation during the first few days that they are lying in hyperextension to mask the pain due to the mechanism described. If this is done promptly and adequately, respiratory movements will be inhibited only slightly and the tendency to hypostatic pneumonia will be greatly reduced.

Abdominal distention may occur during the first few days after an injury of the spine due to paralysis of the autonomic nervous system. It should be treated vigorously along the same lines used in treating paralytic ileus following abdominal operations. Passage of a Miller-Abbott tube into the upper alimentary tract is the best method. Pitression (1 cc. ampule) or prostigmine (1 cc. ampule) may be given intramuscularly. Rectal tubes should be used. Enemas of soap suds, water, or water and glycerine may be tried. Hot stupes to the abdominal wall are often effective.

Decubitus Ulcers.—All so-called "bed sores" are pressure sores and are invariably the result of local ischemia caused by continued pressure upon soft tissue. It is not correct to regard pressure sores as "trophic" disturbances. They occur with special frequency in regions where bony prominences are directly covered by skin; the sacrum, trochanters, heels, ischial tuberosities, knees, and anterior superior iliac spine are the most common sites.

There are engorged venules, thrombosed smaller veins and thickened arterioles in decubitus ulcers. Capillary thrombosis manifests itself by the absence of blanching of the skin on pressure. A loss of 50 gms. of protein per day as a result of drainage from these ulcers has been reported and the role of hypoprotinemia in their development has been emphasized as have vitamin B-complex and vitamin C deficiencies.[1, 2]

Prevention is the best treatment. This demands constant vigilance to prevent continuing pressure by a hard bed, or by other parts of the body, upon soft tissues overlying bony prominences. Frequent changes of position in bed, at least every 2 hours; the use of Stryker frames attached to the bed, a scrupulously clean and dry skin; alcohol rubs and the application of cocoa butter to the skin; smooth, unwrinkled bed linen; a firm pillow, or a folded blanket, of sufficient thickness placed crosswise beneath the calves of the legs so that the heels do not touch the bed; small pillows or cotton pads placed between the knees and between the ankles and toes; and attention to

[1] Poer, D. H. [illegible] ralyzed Patients Due to Wartime Int[illegible] 1946.

[2] Mullholl[illegible] and Shafiroff, B.: Protein Metabolism and Bed Sores, Ann. Surg., 118, 1015, 1943.

a proper diet to prevent anemia and hypoproteinemia are some of the factors which must be vigilantly enforced by doctors, nurses, and attendants. In turn, the patient must be made conscious of his part in the prevention of decubiti.

A fairly large percentage of decubitus ulcers will heal under conservative treatment if the patient's nutrition is correct, if nitrogen balance is maintained and if anemia and hypoproteinemia are avoided. However, certain conservative surgical measures are also necessary. All devitalized skin, fat and subcutaneous tissue must be excised. Wet saline compresses, Dakin's solution, sulfonamide ointment, penicillin jelly, chloraqua, infrared-ultraviolet rays, thyrotricin have all been used locally to stimulate the production of healthy granulation tissue. It is wise to convert the open ulcer area into a closed wound as early as possible after the bed is fresh and clean Many procedures have been devised for operations in typical areas, but the method of choice must depend upon the surgeon who should decide whether a procedure with resection of bone in successive stages, rotating flaps, delayed flaps, or wide undermining with sliding flaps will best suit the individual case.

TREATMENT OF GENITO-URINARY CONDITIONS

The treatment of genito-urinary complications is a major problem in every paralyzed patient. Urinary infection alone will prevent a return of function in the spinal cord and is the most common cause of death.

Traumatic Cord Bladder.—The neurogenic bladder may be defined as a bladder malfunctioning because of a derangement in the normal physiological nervous mechanism which controls the emptying and filling of the organ. Because of the disruption of this mechanism in injuries of the spinal cord, one is faced with various types of malfunction. There may be complete incontinence or retention with overflow.

The forces which expel the contents of the bladder are the detrusor and abdominal muscles. The forces of retention are the muscle bundles which form the vesical neck and the striated muscles forming the pelvic diaphragm, of which the external sphincter is a part. Relaxation of the sphincter depends upon the anterior perineal muscles as well as upon the vesical neck.

It is generally agreed that the bladder mechanism is innervated by three sets of nerves (1) fibers from the sympathetic system, arising from the first three lumbar sympathetic ganglia and reaching the bladder through the presacral nerves and the hypogastric plexus This pathway is thought to inhibit the contraction of the detrusor muscle and to cause the contraction of the

internal sphincter. It is sometimes called the "filling nerve." (2) Fibers of the parasympathetic system derived from the first four sacral levels reach the bladder through the nervi erigentes and the hemorrhoidal plexus. This pathway is antagonistic in function to the sympathetic pathway. Stimulation of the parasympathetics causes strong contracture of the detrusor muscle and a relaxation of the smooth muscle of the urethra. (3) Fibers of the spinal system derived from the third and fourth sacral roots reach the perineum through the internal pudendal nerve and innervate the striated muscle of the external vesical sphincter. It is thought that the balance between the functions of the sympathetic and parasympathetic nerve supplies of the bladder is the foundation of the normally functioning viscus and that the somatic nerve supply enables one to control voluntarily the act of urination.

It must be remembered that the innervation of the bladder is all derived from the lower lumbar and sacral levels of the cord and that theoretically then there may not be so much interference with bladder function in cord lesions above this level except for the loss of voluntary control over voiding. Practically, this works out in a satisfactory manner in handling the bladder disturbances of these patients. Centers in the sacral region of the cord below the site of the lesion keep up a slight tonus in the detrusor, and apparently give rise to an automatic voiding reflex when the bladder becomes distended. While much weaker, perhaps, than the normal voiding reflex, this reflex may be increased by stimulations reaching the cord below the level of the lesion. This accounts for the emptying of the bladder as part of the mass reflex excited in some complete lesions of the cord by pinching or stroking the legs

The purposes of treatment of the paralyzed bladder are twofold; (*a*) the prevention of serious sepsis of the urinary tract, and (*b*) the preservation of normal bladder capacity and musculature. The former is necessary for the preservation of life, but the latter is very important if the patient is to make a satisfactory social adjustment after his recovery from the acute phases of his injury.

In order to assess the condition of the bladder after a spinal cord injury, it is necessary to make a series of examinations which must be correlated to yield an accurate diagnosis. These include cystoscopy, testing the bulbocavernosus reflex, urography, cystography, sphincterometry, cystometry and a determination of the bladder capacity and the residual urine. Bors[1] has described the purposes and advantages to the care of the urinary bladder of each of these procedures and emphasizes that they should be made repeatedly and at regular intervals because the status of a cord bladder changes from time to time

Obviously, the completeness of the cord lesion and its level play a major role in the degree and date of recovery of bladder function.

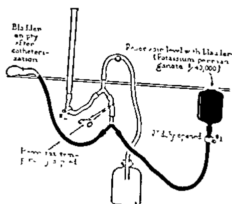
[1] Bors, Ernest Spinal Cord Injuries, Veterans Administration Technical Bulletin, TB10-503, 1948

In his study of 339 patients, Bors found that function was restored in 91 per cent of the patients with lesions above the conus medullaris and 97 per cent of those whose lesions were below the conus. In the civilian patients, function returned within ten months in 80 per cent of the cervical cord lesions; 78 per cent of thoracic and 73 per cent of those lesions below the 12th thoracic segment. An additional 20 per cent of cervical; 22 per cent of thoracic and 18 per cent of lumbosacral injuries recovered bladder function after more than ten months. There was a pronounced difference in the recovery of bladder function in patients with war injuries. The slower recovery was believed to be due to the fact that initial suprapubic drainage prolonged the period of imbalance in the bladder. We observed in our military patients, only 14 per cent recovery in the cervical, 34 per cent in the thoracic and 16 per cent in the lumbosacral injuries.

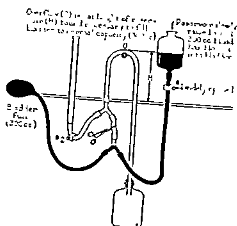
The goal of management of the bladder is to prevent distention and over-stretching of the bladder muscles, infection, and restore function. To allow the bladder to overflow is a poor method because it permits overdistention. Manual expression is contraindicated in the presence of cystitis and may be followed by rupture of the bladder wall. Intermittent catheterization should not be carried out for more than forty-eight hours because of the danger of infection. Suprapubic cystostomy was practiced routinely during World War II but it neither guaranteed dryness nor safety against infection or stone formation Most of the cystostomy wounds were placed wrongly, and there was leakage about the tube. In many of the cystostomies which were made too low, an osteomyelitis developed. We believe, therefore, that a No. 16 F Foley bag catheter should be introduced into the bladder and connected with a closed sterile drainage system.

There are several acceptable forms of instituting tidal drainage of the bladder but we have found the system illustrated in Figure 127, as described and used by Scarff[1] to be satisfactory. It is important that the catheter and drainage tube should be brought out over the thigh rather than allowed to fall down between the thighs and lie along the bed In this latter position an acute angulation of the urethra occurs at the level of the suspensory ligament which will contribute seriously to the production of irritative urethritis. The in-lying catheter should be removed approximately once a week. This should be done in the morning and a new catheter should not be replaced until late in the day This allows eight to ten hours for voluntary micturition to occur if the bladder mechanism is ready to assume that function. If voluntary micturition does not occur, the

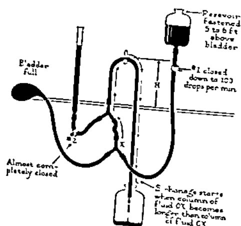
[1] Scarff, John E The Surgical Treatment of Injuries of the Brain, Spinal Cord and Peripheral Nerves Surg., Gynec and Obst., 81, 405, 1945.



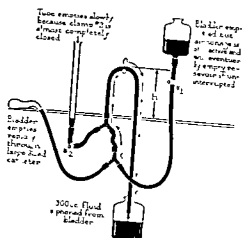
A



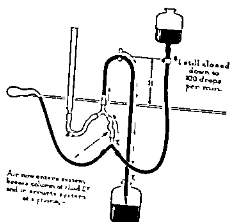
B



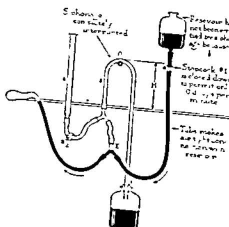
C



D



E



F

FIG. 127.—Successive steps in the operation of a successful tidal drainage set. (Courtesy of Dr. John E. Scarff and Surg., Gynec. and Obst.)

catheter should be replaced and tidal drainage continued for another week, when this procedure should be repeated.* The tidal drainage should not be discontinued without good and specific cause before voluntary control of the bladder returns.

The employment of exercises to condition the voiding reflex, drugs, presacral neurectomy, pudendal neurectomy, vesical neck block, pudendal nerve block, sacral nerve block and transurethral resection are all methods which may be useful in specific instances to help the patient gain recovery of bladder function.

Infection of the Urine and Calculosis.—The reaction of the urine is usually alkaline due to the infecting type of microorganism and attempts to change the reaction by the administration of ammonium chloride, mandelic acid or an acid-ash diet are usually unsuccessful. Impaired function of the renal tubules of an infected kidney adds to the alkalinity of the urine. The proper care of an irrigating-drainage system helps prevent infection and the accompanying attacks of headache, nausea, vomiting, chills and fever. Any mechanical obstruction in the genito-urinary tract must be excluded by cystoscopy and ureteral catheterization. In mild cases, calcium mandelate may be used but the administration of the sulfonamides, penicillin or streptomycin is more effective depending upon the identification of the organism and its sensitivity to the drug in question.

The formation of urinary stones is a frequent complicating problem. Improper drainage, infection, hypercalcinuria caused by immobilization, and urine alkalinity may all precipitate the formation of calcium and magnesium phosphate and carbonate stones. Vesical calculi are treated best by crushing as soon as the stone is recognized. In the upper urinary tract, stones may be treated conservatively unless an obstruction becomes complete.

Periurethral Abscess and Epididymitis.—An ulcer of the urethra at the acute angle of the penoscrotal junction may be the forerunner of a periurethral abscess The use of a catheter which is larger than a No 16 F is the most common cause of the ulcer Often the abscess develops weeks after the catheter has been removed and may become a large scrotal phlegmon very rapidly. The abscess should be incised, drained and the penoscrotal angle straightened by strapping the penis to the abdomen

* The tidal drainage sets may be filled with Suby's solution which consists of citric acid (monohydrate) 32 3 grams, magnesium oxide (anhydrous) 3 8 grams, sodium carbonate (anhydrous) 4 4 grams and distilled water to 1,000 cc. This solution has a pH of 4 and dissolves soft calcium phosphate stones and is mildly antiseptic A variation of this solution has a pH of 5 and contains double the amount of sodium carbonate but is less irritating. Distilled water is alternated with this solution in the set

Epididymitis is a frequent complication in the patient with a spinal cord injury and may occur at any time or stage in his treatment. The genitalia are easily traumatized by pressure during sitting if they are not supported properly. The application of an icebag, support and antibiotics should be instituted promptly. If an intraurethral catheter is in use, it should be changed every 24 hours. Incision and drainage are rarely necessary if careful conservative treatment is carried out.

Sexual Function.—The disturbances of sexual function in the patients we have studied with incomplete cord lesions vary from a condition which is almost normal to impotency. Reflex erection and reflex ejaculation with genital anesthesia is a rare occurrence. Reflex erection has persisted even when complete destruction of the cauda equina has occurred, so that the reflex arc apparently need not pass through the cord but only through the parasympathetic ganglia close to the genitalia. Bors has reported a return of erection in 86.6 per cent of a group of 90 patients with incomplete and 67 with complete lesions at various levels of the spinal cord. With lesions below the seventh thoracic segment, erections returned but ejaculations and orgasms occurred in less than 20 per cent of the patients. In another study it was found that erection on local stimulation as compared with psychic stimulation was 6 times as common in complete lesions and about equally common in incomplete lesions. Bors explains the survival of erection in the absence of ejaculation by the fact that the latter depends on two separate reflex arcs, whereas the former depends on a simple single reflex arc.

GENERAL NUTRITION

Pollock and his associates have made a detailed study of various important components of the general bodily state in patients with spinal cord injuries. A constant finding was a mean weight loss of 31 pounds which was not regained. The basal blood pressure was found to be low in cervical cord lesions and there was no increase proportionate to age. In these lesions, orthostatic hypotension was noted most often. A defect in the concentration of urine as well as in the day-night ratio was found although no signs of kidney disease were noted. Glucose tolerance curves were abnormal, although there was no evidence of disease of the liver, pancreas, adrenal glands or thyroid. Hunger and thirst were experienced as well as a sensation of fullness after eating. Nausea was produced by all the various appropriate stimuli. The water intake was increased particularly in cervical cord lesions.

The loss of protein results in a negative nitrogen balance in a large number of patients and as has been said, drainage from decubitus ulcers contributes up to 50 gms. of protein per day. This loss must be balanced by a high protein diet or by supplemental parenteral administration of protein-hydrolysates, plasma and blood. Blood transfusions should be used to keep the hemoglobin level above 80 per cent of normal, the red count at 4,000,000 or more and the total blood proteins at 6 gm. per 100 cc.

A high vitamin intake and maintenence of a satisfactory carbohydrate balance are also necessary. Overfeeding with a high caloric intake may be desirable in the early stages of the care of these patients.

Disturbances of calcium deposition are quite common and result in erosive bone lesions and calcification of the soft tissues, particularly in the vicinity of decubitus ulcers over the trochanters and ischial tuberosities We have observed several fractures of the femur which presented difficult problems in treatment and callus formation.

Bowel Habits.—In 91 per cent of 244 patients, studied by Pollock, a change from dependence on enemas to reflex defecation was effected. This averted the untoward effects of enemas, such as excessive sweating, nausea, spasms, weakness and fainting. Reflex defecation may occur in a patient with a complete lesion of the cauda equina and is apparently mediated by a local nerve plexus in the wall of the rectum. Stretching of the rectum, or the presence of a mass, is an adequate stimulus for contraction of the rectum and relaxation of the sphincter. The tonicity of the sphincter muscle has no apparent relation to successful development of reflex defecation. To change from the use of enemas to reflex defecation may take only a few days and enemas should be discarded early after injury. Impaction of feces should be avoided by the administration of lubricants. The patient should be placed in a proper sitting position during defectation in order to make the best use of his remaining innervated abdominal muscles and diaphragm and he should be taught how to exert pressure on his abdominal wall with the palms of his hands if his abdominal musculature is completely paralyzed.

RELIEF OF SPASMS

Abnormal reflex activity, appearing early or late, may manifest itself in the responses to plantar or other superficial stimulation, to muscle stretching, to genital stimulation and to the stimulation of either the bladder or the bowel. The vasomotor, pilomotor or sudomotor responses which accompany the various striated muscle contractions may be entirely erratic. Certainly, the patterns of

reflex responses are rarely repeated often enough to make them reliable diagnostic or prognostic agents.

Soon after injury, and the state of areflexia, primitive defense reflex mechanisms develop in the isolated distal segment of the spinal cord. These primitive patterns then change from simple withdrawal responses and flexor posture to the increasingly complicated movements observed in the pseudospontaneous spasms and in exteroceptive reflexes. The reflex response rarely consists of a simultaneous contraction of all participating muscles; rather, it is the result of successive reflexes elicited by a single stimulus. So-called spasticity is the resultant of many reflexes—stretch, myotatic, body and exteroceptive reflexes. The frequency of occurrence of paraplegia in flexion, spasticity, pluck reflexes, pseudospontaneous spasms and the potential total activity of the spinal cord is related to the level of the lesion, the incidence diminishing as lower segments are reached.

Some patients never have anything more than the mildest degree of spasm and in others the deformity produced by heightened reflexes requires surgical treatment. We have observed that spasm has been greatest in those patients in whom early care was incomplete, and an early state of sepsis and malnutrition was established.

In general, the use of common sedatives has not affected the heightened reflexes. Curare in its various forms has been used upon a considerable number of patients but a prolonged effect and the avoidance of undesirable side effects could not be attained. Posterior rhizotomy, as well as the intrathecal injection of alcohol, were used without effect in some instances among our group of patients before they came under our care. Anterior rhizotomy as high as the tenth thoracic segment has been uniformly successful when a sufficient number of segments were exposed but we have studiously avoided this operation until it was established beyond doubt that the patient could anticipate no further recovery. The scarred proximal and distal ends of the severed cord have been freshly resected with the local removal of all irritating factors, such as abnormal vascular [illegible]

[illegible]

Some patients, suffering predominantly from adductor spasm, and in whom the tendency for the lower extremities to cross has been a real impediment to their otherwise possible ambulation, have had immediate relief with an improvement in their use of canes, crutches, braces, or wheelchairs, following the bilateral section of the obturator nerves, as approached through the lateral reaches of the space of

[1] Scarff, J. E., and Pool, J. L.: Factors Causing Massive Spasm following the Transection of the Cord in Man. Jour. Neurosurg., 3, 285, 1946.

Retzius. In some individuals muscle tenotomy must be performed to relieve the tendon contractures which have occurred.

RELIEF OF PAIN

Root pains may occur in lesions of the spinal cord at any level due to the pressure exerted by a foreign body or by bone callus formation upon the roots. They may be relieved by direct attack upon the offending compression or in cauda equina lesions if several roots are involved, a properly performed cordotomy is indicated. Burning and tingling pain which does not follow the distribution of spinal segments, which may appear immediately or months after the injury, is not benefited by any surgical procedure in our experience. These patients should be treated psychologically, and their nutrition improved, and occupational therapy, education and recreation facilities provided. Some patients complain of pain which represents the functioning of autonomic mechanisms; fullness in the abdomen, vague periumbilical pains, flushing, nausea, sweating and headache are the common symptoms of complaint. These complaints also tend to disappear as the injured patient becomes more adjusted and his sensation of frustration and bitterness disappears.

REHABILITATION

The psychological depression which follows an injury to the spinal cord varies with each individual patient but must be corrected before any hope for successful rehabilitation can be entertained. The psychological reaction of the patient with a spinal cord injury and his paralyzed extremities differs from that of the amputee who sees a material defect quite easily. The former, however, sees his limbs still present and is encouraged thereby to entertain false hopes.

It may require a year or more for the patient's pretraumatic personality to show itself Extroverts and individuals with stable family relations adjust more easily but in all, a period of dependence upon the assistance of others leads to frustration and immature emotional reactions Disappointment and bitterness are often directed against the doctors and nurses because of their failure to cure the injured spinal cord The medical, nursing, rehabilitation staff and the family must cooperate to bring about as rapid an adjustment as is possible

Physical Therapy. - The purpose of physcial therapy is to prevent contractures and deformities, to increase muscular function by strengthening weakened muscles and re-educating unparalyzed or less involved muscles The extent to which all of the forms of physi-

cal therapy can be used by the patient depends upon whether or not he is in an institution in which physical therapy is given to a large group, or is a single patient at home where appliances can be added, but only within the limits of the living requirements of relatives.

Breathing exercises should be taught immediately to improve the vital capacity of the lungs while at bed rest and to help in gaining relaxation in the future days of increased voluntary or reflex activity. Passive movements should be started early and proceed to active and resistive exercises designed to carry out the full range of the particular movement. As has been pointed out, passive or active stretching forces lymph, and so presumably, metabolic products from the muscles and they have a distinct influence in retarding the formation of connective tissue, which begins rather quickly in lower motor neuron lesions. Part of the late contractures in flaccid paralysis may be due to the shrinkage of this newly formed tissue which is soft and extensile. These therapeutic movements also increase the range of motion in an already stiffened joint and help to keep a mobile joint active so that when the time comes it is ready to perform its part as an effector mechanism. These exercises should be carried out slowly, gently, and never with quick, jerky movements. Each separate passive exercise should be individualized, and the patient be required to make the attempt to perform the movement simultaneously or to attempt to hold the part in the position imposed upon it.

Often, in the patients with spasms, these active and passive movements are better carried out under water and a Hubbard tank or a therapeutic pool should be made available to the patient. The warm water eliminates the spasms, the effects of gravity and relaxes the patient. Rubber rings aid the patient to float in the pool; the patient feels secure, co-ordinated movements can be developed; muscles strength in uninjured muscles can be increased and psychologically, the patient is able to move in three dimensions without outside aid and achieve a goal equal to a normal individual.

Gentle massage, consisting of stroking rather than rubbing movements, improves the venous and lymphatic circulation in the muscles Later, more pressure may be used than at the beginning, but care should be taken not to injure the paralyzed muscles by compressing them against bones Massage should be preceded by exposure of the extremities to heat for from twenty to thirty minutes. Radiant-heat, the infra-red light, hot packs or a whirlpool bath of warm water are all useful, but they must be used with extreme care because of the ease with which it is possible to burn the skin of a denervated area

The use of electrotherapy in patients with heightened reflexes and spasms accomplishes very little as compared with the results which may be obtained in a patient with flaccid paralysis. In upper motor neuron lesions, muscle atrophy does not occur except as the result of non-use. Since the lower motor neuron is uninjured, the trophic nerve supply to the muscles is intact, and the intelligent employment of massage and active and passive movements will accomplish more with far less danger of harmful results. On the contrary, in flaccid paralysis, electrotherapy is indicated to prevent the atrophy of muscles and fibrosis, to increase nutrition, and to conserve the functional capacity of paralyzed muscles until sufficient return of function has taken place to permit of active motion.

It is necessary to understand clearly the method of action of treatment by electricity. Stimulation by an electric current of sufficient strength produces a contraction of the muscle. It is this active contraction which conserves the volume and nutrition and keeps the muscle fibers in a functional state adequate for voluntary movement when regeneration occurs. The only requirement of electrotherapy is that it produce a contraction of the paralyzed muscle. This cannot be obtained by the faradic current because the duration of each stimulus is too short in relation to the changed chronaxie of the nerve and muscle. Therefore, a galvanic current must be used. It can be used in its simplest form of a continuous current, or in the form of sinusoidal currents of various wave types. Galvanic continuous current is often painful, and therefore, other types of waves are useful in children or sensitive patients Because the muscle contraction occurs only upon making and breaking the current, prolonged stimulation is unnecessary. The current should be applied with short makes and immediate breaks of the current by the use of a suitable electrode.

During the first few months following the paralysis the muscles are hyperirritable. Unipolar stimulation will produce contractions in the muscles most affected by the weakest currents because of longitudinal stimulation. Consequently, fatigue in these muscles must be avoided. It must be emphasized that deep muscle sense may be defective, and the patient is unable to tell when his muscles are fatigued.

Later the most affected muscles become less irritable and unipolar stimulation produces a spread of current to unparalyzed muscles which alone may contract to the injury of the patient Therefore, bipolar electrodes should be used at this stage. Although polar inversion is not always the rule in degenerated muscles, it is sufficiently the case so that the positive pole should be used as the active electrode. It is also the least painful.

In producing contractions of the paralyzed muscles, care must be used to prevent the force of gravity acting against the contracting muscles. This factor may hide movements of segments about the joints and may increase the onset of fatigue. The extensors of the foot may not produce a movement with the extremity in a position of foot-drop, but if the patient rests the leg on a board at the outer surface, dorsal flexion may occur.

Contraction of muscles produced by electric current is a valuable aid to re-education and active motion. Although the patient may not be able to produce dorsiflexion voluntarily, the ankle may be held in that position after electrical stimulation has produced dorsiflexion.

Care must be exercised in treating areas of skin in which sensation has been destroyed. Burns are produced easily, and when ulceration of one occurs, healing is difficult.

When the nerve and muscle have recovered sufficiently to contract to faradic stimuli, both galvanic and faradic stimulation are advisable, the latter to produce more prolonged or tetanic contractions.

Splints.—Much can be done by the intelligent use of splints in patients with a flaccid paralysis. While the patient is in bed, foot-drop may be avoided by the use of sandbags against the soles of the patient's feet and a cradle over the legs to keep the weight of the bed clothes off the feet. These require careful and constant adjustments, and it may prove more desirable to use light crinoline posterior molded splints which may be removed easily for massage and passive movements. Adhesive-tape straps from the sole of the foot to the dorsum of the leg may be effective also.

When the patient becomes ambulatory, the most satisfactory splint to prevent foot-drop is one patterned after that shown in Figure 128. This is simple and can be attached to the shoe easily. The remainder of the extremity also requires support in the majority of patients. Various types of braces of light materials have been devised but the common principle consists of long, walking calipers or stirrup braces.

Corrective Therapy—Every possible method must be used to improve muscle power in those muscles of the body which are weak or uninvolved, in preparation for making the patient ambulatory. Dumbbells, bar-bells, springs, and pulley weights should be used. The arm flexors and extensors, finger flexors and thumb flexors, dorsiflexors of the wrist and the shoulder girdle muscles must be developed for crutch ambulation. Mat exercises and training exercises in getting from a wheel chair to the floor and back again must be practiced. All types of games and sports should be modified and used

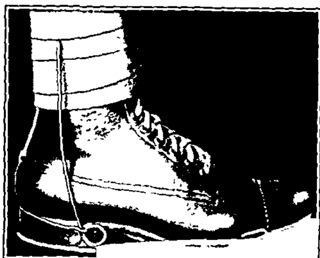


Fig. 128.—A satisfactory and simple splint for the correction of foot-drop deformity. (After Buerki.)



Fig. 129.—Muscle exercises in a tank of water. This method removes friction and weight from the limbs

to avoid boredom and discouragement and to develop a sense of accomplishment.

Ambulation.—It is obvious that the degree of ambulation possible in any given patient depends entirely upon the level of the injury to the spinal cord, and it also determines the type of gait which is acquired. Success in ambulation, as far as the patient is concerned, depends upon how closely this approximates normal walking. One must avoid the sweeping assurance to the patient that he will walk again, when only ambulation is meant; the patient makes no such differentiation in his mind.

Exercises of balance between parallel bars or in a walker are the basis for all ambulation and they are followed by leg-swinging exercises, and eventually side, back, and forward stepping exercises. We believe the walker is an excellent support because it imitates very closely the motion which is needed when the patient is able to use crutches. When the patient graduates to crutches, he must be taught balance again. Then, depending upon the level of his lesion he may learn to use a four-point gait or may be restricted to a tripod gait. In the former, which patients with lesions below the thoracic tenth segment can learn, the patient advances one crutch and then pulls the contralateral leg forward. This is repeated successively on each side with great effort being exerted from the shoulder girdle. This type of ambulation simulates the normal gait. In tripod walking, the patient places both crutches forward simultaneously and then swings his legs forward until his toes lie near a horizontal line which connects the crutch tips on the floor, or beyond that line.

Instruction in ambulation must include walking on uneven ground, stairs, curbs; getting in and out of wheel chairs from beds, toilet seats, automobiles unaided; sitting down in any chair; getting in and out of the bath tub; putting on clothing and in short, becoming independent of outside assistance. One of our patients with a cervical spinal cord injury received in a diving accident, had the most complete care in his home. However, it was not until he graduated from high school and went away to college where there were other similarly handicapped individuals that he acquired what he terms, "real independence." It has been our experience that most patients with high spinal cord lesions, eventually give up attempts at ambulation and are completely adjusted to wheel chair mobility, particularly because the effort to ambulate is so great and the result does not simulate a normal gait sufficiently to satisfy them.

Social and Economic Rehabilitation.—The primary object of occupational therapy is to divert the patient, but it can be used to develop habits of work and to provide purposeful and constructive activities which improve the patient's muscle coordination and di-

minish his mental and emotional stress. All types of feeding and writing instruments have been devised particularly for patients with quadriplegia. The continuation of school activities for younger patients and the extension of education in older patients should be undertaken with the intiative coming from the patient. Manual arts therapy in radio repair, printing, photography, drafting, blueprinting, watch repairing and machine, wood and plastic shop training are some of the possible outlets for these patients.

Finally, it must be remembered that patients with spinal cord injuries are individual problems and they need individual attention from their doctor. Most important of all, it must be remembered that they need total medical attention and care, regardless of the specialty category to which their own physician belongs, but correlated by one doctor to whom they may turn for the answers to all of their questions. This responsibility to these patients cannot be shared. This attention and care must be available to them for the remainder of their lives and they must be taught to seek follow-up examinations at regular intervals.

FRACTURE-DISLOCATIONS OF THE SPINE WITHOUT CORD INJURY

Often the vertebræ may show surprising injuries without the slightest evidence of damage to the spinal cord. In many fracture-dislocations, the rotation of one vertebra upon the other occurs in the long axis of the spine, and although roentgen-ray films may give the impression of a severe displacement, the spinal cord may not be injured. On the contrary, far less bony damage may be accompanied by severe angulation which produces tremendous cord damage. It is in the treatment of these fractures or dislocations that injury to the cord may occur and yet this possibility is so frequently disregarded.

A minor blow on the chin or occiput with the head rotated may cause a unilateral dislocation of the first two cervical vertebræ, with or without a fracture. Because of the horizontal position of the articular processes and a rupture of the capsular ligament, the *articular facet of the atlas may slip forward over or on to the anterior* marginal lip of the facet of the axis. The head remains fixed in a rotated position and any attempt to move it causes extreme pain. Often this condition may be produced by accidental violence so minor in character that the patient's physician fails to recognize the possibility of its presence We have observed one such instance, but the patient came to our attention eight weeks after the dislocation, when any attempt to reduce the deformity was bound to be difficult.

Bilateral dislocation and fracture-dislocation of the cervical vertebræ constitute the most serious injuries of the spinal column because the incidence of injury to the spinal cord is so high. When fractures complicate the dislocation, the extreme danger of increasing the spinal cord injury, or producing injury if none exists, by manipulation will give the surgeon many anxious moments. We feel sure that no dogmatic rule can be laid down to cover every case, but that the character of the spinal cord lesion as judged from the symptoms, the presence or absence of spinal subarachnoid space block, the roentgen-ray appearance of the bony damage, and the clinical condition of the patient must all be carefully weighed before a decision is made as to the method of treatment.

Alfred Taylor[1] strongly recommended immediate reduction of this type of injury by traction and manipulation. McKenzie[2] and Crutchfield[3] have devised a method of traction by the introduction of tongs into the skull which are attached to weights through a movable pulley at the head of the bed. By use of pads placed under the head and neck, it is possible to change the direction of the pull or at any time additional manipulative acts can be carried out. Tong traction is far more efficient than pulling upon a halter about the chin and head; it is free from the dangers associated with manual reduction; the force can be accurately controlled, and the application of the tongs through small scalp incisions under local anesthesia is quite painless. There is no doubt that this method may lessen the necessity for laminectomy in fracture-dislocations of the highest cervical vertebræ, particularly when a subarachnoid space block is absent. Repetition of the Queckenstedt test is an excellent means of checking the efficacy of this method of traction if a block exists in the beginning.

The tongs are applied transversely to the vertex of the skull in a vertical plane which passes through the articulations of the cervical spine. The mastoid tips are within this plane and serve as satisfactory landmarks. The Crutchfield tongs are designed so that the traction bar can be used as a guide for the correct placement of the tong points into the skull. The traction bar is turned down and placed against the scalp with the arrow pointing to the midline of the skull. The tong points are lowered to the scalp and the points of contact are marked with dye to indicate the proposed stab wounds, which are made under local anesthesia.

Perforation of the outer table of the skull is made with a 2 mm. drill point which goes in 3 mms. The tong points are fitted in and made secure by adjust-

[1] Taylor, A. S. Fracture Dislocation of the Cervical Spine, Ann. Surg., 90, 321, 1929.

[2] McKenzie, K. G. Fracture, Dislocation, and Fracture-Dislocation of the Spine, Canad Med. Assn Jour, 32, 263, 1935

[3] Crutchfield, W. G. Further Observations on the Treatment of Fracture Dislocations of the Cervical Spine With Skeletal Traction, Surg., Gynec. and Obst., 63, 513, 1936

ing thumb screws. Collodion dressings should be applied and changed frequently. The head of the bed is elevated 12 inches and 5 to 10-pound weights should be attached in a direction depending upon the individual patient.

When dislocation of the cervical vertebræ is accompanied by definite symptoms of injury to the spinal cord, many surgeons believe that a laminectomy can do no good and may do harm. Reduction and fixation of the dislocation offers the best chance for restoration of function to the spine, nerve roots, and cord. With this viewpoint, in certain cases, we are in entire accord but any manipulative method in the presence of a fracture may result in irreparable damage to a cord which has previously suffered only a physiological interruption of its function.

A very large number of injuries to the thoracic and lumbar spine result in compression fractures. A small ledge of bone may be pinched off in front of the vertebral body, or the anterior surface of the body may be crushed so that a wedge-shaped vertebra with its point toward the ventral surface results. Not infrequently very little deformity is produced, and many of these injuries are regarded as "sprains." Lateral roentgen-ray films will show an irregularity in the outline of the vertebral body without any change in the width of the intervertebral spaces, which may not be demonstrable upon the antero-posterior films.

As Magnuson[1] has pointed out, the objects of treatment of compression fractures should be: "(1) the reduction of the deformity; (2) the maintenance of reduction; (3) if reduction is impossible, the establishment of compensatory curves to allow the spine to assume an upright position; (4) the repair of damaged and over-stretched ligaments and muscles; (5) the prevention of abnormal strain on ligaments and muscles at a distance from the fracture."

In a general way, a safe principle of treatment to follow is to reverse the mechanics of production of the fracture. But, before any procedure is instituted, fractures of the articular processes must be ruled out, else a dislocation may occur and pressure upon the spinal cord may result Methods of producing hyperextension of the spinal column may utilize automobile jacks placed beneath the mattress of the bed; the Rogers' frame, or the method of A. G. Davis[2] Late treatment in these cases consists in immobilization of the spine in the upright posture by a Taylor spinal brace. Spinal bone grafts or fusion operations have been employed when relief of symptoms and restoration of function cannot be obtained by these non-operative methods.[3] (Fig. 130.)

[1] Magnuson, Paul B Fractures, Philadelphia, J. B. Lippincott Company, 1933.

[2] [illegible] Surg. 11 [illegible] 1929

[3] [illegible]

Fractures of the spinous processes, usually produced by a direct blow, may be accompanied by a fracture of the laminæ. There is little or no displacement of the fragments because of the firm muscle fixation upon each side of the process and the strong posterior longitudinal interspinous ligament. Fractures of the articular processes rarely occur without serious damage to other portions of the spinal

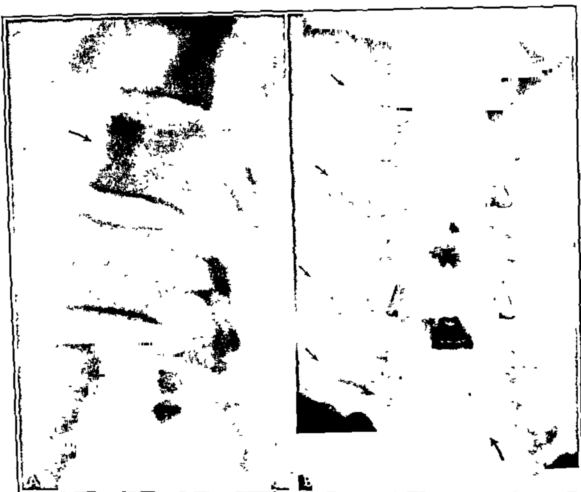


FIG 130.—(*A*) Compression fracture of the second lumbar vertebra without spinal cord symptoms. (*B*) Multiple fractures of the transverse processes of the lumbar vertebræ and a fracture of the fifth lumbar vertebra. The patient was entirely free of spinal cord symptoms.

column in the immediate vicinity. Fractures of the transverse processes occur only in the lumbar vertebræ and are frequently observed in large industrial surgical centers. Immobilization in a plaster cast should be immediate, and the patient should be kept in bed for six to eight weeks. Fibrous union takes place between the fractured surfaces and since non-union is shown by the roentgen-ray films, false corroboration for the patient's claim that he has pain

and is unable to work is present. Determination of the degree of disability in these latter instances is often required of the surgeon before compensation boards and accurate examination of the points of tenderness in these patients upon lateral, forward, backward, and rotary movements of the spine requires patience and carefully checked and often repeated observations.

CHAPTER VIII

TUMORS OF THE SPINAL CORD

ALTHOUGH the ancients were acquainted with fracture-dislocations of the spinal column and the consequent injury suffered by the spinal cord, it was not until the eighteenth century that a description of compression of the spinal cord by a tumor appeared in the literature.

In the afternoon of June 9, 1887, at the National Hospital for the Paralyzed and Epileptic in Queen's Square, London, Sir Victor Horsley performed a laminectomy on an army officer, aged forty-two years, with spastic paraplegia in extension, who had suffered from severe pain below the inferior angle of the left scapula for three years. The patient had been under the care of Sir William Gowers who had diagnosed a spinal cord tumor and advised operation. In this Sir William Jenner, who had also seen the patient, had concurred. Throughout the operation the carbolic spray was used and instruments and sponges were immersed in 5 per cent carbolic solution. No tumor was found, and the operation was about to be concluded when Mr. Charles Ballance, who was assisting, urged Horsley to go higher. In his report of the case Horsley remarked:

> "I removed another lamina at the upper part of the incision. On opening the dura mater, I saw on the left side of the subdural cavity a round, dark, bluish mass about 3 mm. in diameter, resting upon the left lateral column and posterior root zone of the spinal cord. I recognized it at once to be the lower end of a new growth and therefore quickly cut away the major part of the lamina next above. This enabled me to see almost the whole extent of the tumor when the dura was divided "[1]

Tumors of the spinal cord are not as common as intracranial tumors, but the symptoms which they produce are so pronounced and the successful results of surgical treatment are often so dramatic and striking that they deserve careful attention. Though in our own series we have 1215 verified intracranial tumors, our series of verified tumors of the spinal cord number only 181.

Tumors within the spinal canal can be divided into those which are extradural or intradural, and the latter group may be extra-

[1] It is interesting to note that although the cord was swabbed with a 5 per cent solution of phenol and the patient had a cerebrospinal fluid fistula for six weeks, he made an astonishingly complete recovery, and a year later wrote to Horsley that he was in excellent health and working sixteen hours a day.

Laminectomy had been described as an operation not within the range of practical surgery and Horsley collected records of 58 other patients untreated by operation, each of which had been fatal.

medullary or intramedullary in location. This anatomical classification may be very definitely correlated with the different types of tumors and the clinical symptoms which they produce. Spinal cord tumors may be classed further according to their *horizontal location* and vertical level within the spinal canal. The anterior, posterior roots and the dentate ligament make it possible to specify a dorsal, dorso-lateral, ventro-lateral, or ventral tumor; and with these various situations the progression of clinical symptoms vary. In the 181 cases we have encountered, there have been 84 extradural, 63 intradural, and 34 intramedullary tumors Forty have occurred in the cervical, 89 in the thoracic, 31 in the lumbar portions of the spinal cord, and 21 have been tumors of the cauda equina.

Intradural, but *extramedullary*, tumors arise from the sheaths of the spinal nerve roots or from the membranes surrounding the spinal cord. Those tumors which arise from the spinal nerve sheaths are circumscribed, encapsulated growths into a portion of which the nerve root can usually be traced. Usually single, they may grow to an enormous size, particularly in the region of the cauda equina, before they produce symptoms which are easily recognized. However, multiple tumors may be present within the spinal canal as a part of the clinical picture of an advanced neurofibromatosis. Our records contain several of these tumors, which are known as *neurinomas*, and the majority were found in the cauda equina In each patient, however, there were other signs, such as pigmented areas in the skin which neurological surgeons have come to associate with the presence of these tumors elsewhere in the body. The clinical and pathological analogy between the acoustic nerve tumors and these growths is unmistakable. Quite often neurinomas grow along the nerve root, through the dura mater and intervertebral foramen, into the surrounding tissues, but in particular into the thoracic cavity Constricted as they are as they pass through the foramen, they take on an hourglass or dumbbell shape. Microscopically, they show long cells with scanty cytoplasm and a cigar-shaped nucleus A palisade arrangement of the nuclei is very characteristic These tumors do not metastasize and grow very slowly.

The spinal cord *meningiomas*, or *meningeal fibroblastomas*, arise in most instances from the arachnoid membrane, although Elsberg has seen them develop from the inner surface of the dura mater, and we have confirmed that observation. Loosely adherent to the spinal cord, they often fit over its surface like a brake shoe on a wheel. They may vary from a round or oval shape to large, irregular masses which resemble more or less the contour of that portion of the canal in which they are located. Not infrequently dumbbell-shaped

tumors may be found with one enlargement intradurally and the other situated extradurally. These tumors may be located extradurally without any intradural portion, but the majority are intradural tumors and have occurred predominantly in the thoracic portion of the spinal canal. Histologically, the spinal meningiomas closely resemble the meningiomas which are found intracranially and are composed of masses of cells which do not form an intercellular substance. If one looks closely enough, erosion of portions of the vertebra overlying these tumors may be seen. It is Mallory's opinion that the fibroblastomas arise from the arachnoid, but as Elsberg has pointed out not a few are attached only to the duramater. Psammoma bodies are found very frequently in the meningeal fibroblastomas. Just as in the intracranial meningiomas though the tumor is benign, it is necessary to remove its dural attachment to prevent a recurrence. The neurinomas in many instances have an almost identical histological appearance with the fibroblastomas. There is the same whorl formation of cells and the palisade grouping of the nuclei characteristic of both tumors. Mallory would distinguish them by terming the one group arachnoid and the other perineural fibroblastomas

SYMPTOMS

In most cases the onset of symptoms of a spinal cord tumor is very gradual, but the course is progressive. The majority of patients from whom a spinal cord tumor is removed have been treated for various other diseases which might produce pain in the back or legs. Usually it is not until the patient develops a paresis or paralysis that the presence of a tumor is suspected. It is impossible to describe a group of symptoms which would be characteristic of a tumor at each of the vertical and horizontal levels, but it cannot be emphasized too often that the recognition of the symptoms of an upper or lower motor neuron lesion is the first step in simplifying and evaluating the symptoms of a cord tumor.

However, for descriptive convenience we may say that the symptoms of spinal cord tumors may be divided into those which are *irritative* and occur early in the clinical course, and those which are *destructive* and are due to continued pressure and interference with cord function.

Pain in the back, which the patient may describe only as a backache, is one of the frequent early symptoms of a spinal cord tumor. This is particularly true in tumors in the thoracic and lumbar segments of the cord or in the cauda equina. This pain, unlike that of a spinal osteoarthritis, does not disappear upon rest in bed or immobilization. It is made worse by coughing, sneezing, straining at s[illegible]

or any act which raises the intraspinal pressure. In cauda equina tumors the pain is severe and radiates down the posterior surfaces of the thighs so that these cases are often diagnosed as "sciatica." This back pain may be so persistent and severe that the patient assumes various attitudes which will afford relief. Very often palpation or light percussion of one or more spinous processes in the region of the "backache" produces tenderness or even an "electric shock" in the trunk and extremities below that point. Tenderness upon percussion or pressure of the vertebral spinous process is a frequent physical finding in spinal cord tumors regardless of their relation to the cord. The tender spine is not at the level of the tumor but, on the contrary, two or three spines below that point. In other words, the spine of the vertebra which receives its nerve supply from the posterior spinal root of the affected segment is the tender one.

Root pains differ from the back pain just described. They are sharp, excruciating and have a segmental root distribution. They not infrequently are so severe and so localized as to simulate the pain of intra-abdominal disease. This occurred in one of our patients whose gallbladder was removed for right-sided upper abdominal pain. She did not obtain relief until an intradural meningioma in the thoracic segments of the cord was removed.

The radicular pain from involvement of the cervical roots may be so severe that the head and neck are held so rigid that the question of involvement of the bony structures arises. The root pains in the cauda equina or lower lumbosacral cord tumors are often referred to the bladder or rectum as well as to the lower extremities. The pain may be so intense as to require morphine for its relief. It is definitely localized to one certain area of the skin and may be accompanied by a sensation of constriction when the chest or abdomen are involved. Root pains may appear early in the course of a tumor or they may not appear until the growth becomes large enough to involve a posterior root directly. The pains may occur, disappear and recur, depending entirely upon the mechanical change of position which the tumor may undergo. As in the case of back pain, radicular pains may become excruciating upon coughing, sneezing or in any act which increases the intraspinal pressure.

Tingling, burning, numbness, sensation of cold, and various other paresthesias are common early subjective symptoms in spinal cord tumors A zone of the skin corresponding to the sensory roots involved may be very hyperesthetic so that the slightest touch cannot be tolerated.

Whereas subjective *motor symptoms* usually follow sensory disturbances, a patient may first complain of a gradual loss of power in the extremities which often may be described as a feeling of awkward-

ness or stiffness. This motor weakness may begin in one extremity and then involve the other or, not infrequently, it is bilateral at the onset. While the onset of the loss of motor power is usually gradual, a complete paralysis may develop rapidly. This is, however, more pathognomonic of extradural malignant disease in the vertebra.

As the tumor grows and compression of the cord increases, motor and sensory symptoms typical of a destructive lesion occur. Gradually, the lateral pyramidal tracts become involved, and the symptoms of an upper motor neuron lesion develop below the level of the compression. There is a weakness or loss of voluntary motion, spasticity, increased deep tendon reflexes, pathological reflexes, but no muscle atrophy because the primary neuron is not involved. Unless the tumor is situated in the median line anteriorly or posteriorly, the motor symptoms develop first upon the side of the tumor and later involve the opposite extremity. In high cervical cord tumors, spasticity of the upper extremities is always in flexion; whereas, that in the lower extremities may be either in flexion or extension. Rarely, and only if the compression is sudden and severe, a flaccid paralysis may occur due to a certain amount of spinal "shock;" but typically the motor loss is spastic in character. Often the patient complains of the fact that his legs "jump" or "start" at night, and these are in reality flexor spasms which break through the extensor rigidity.

If the tumor is situated on the posterior aspect of the cord, motor symptoms due to pressure at that given segmental level may be unrecognizable. However, if the tumor exerts pressure from the anterior surface of the cord, symptoms of a lower motor neuron lesion may develop. These are characterized by muscle atrophy, loss of deep tendon reflexes, and a flaccid paralysis. These symptoms, together with fibrillary twitchings in the muscles, are more common in intramedullary tumors but do occur in anterior or antero-lateral extramedullary tumors. Consequently, in the same patient with a spinal cord tumor one may see lower motor neuron symptoms at the level of the tumor in addition to upper motor neuron symptoms below that level. It will be apparent that a knowledge of the segmental centers for various muscles and reflexes therefore offers considerable aid in the localization of the tumor. We have removed an intradural neurinoma from the segment of the ninth thoracic level in a patient whose only detectable motor impairment was weakness of the upper half of the abdominal recti muscles. In contrast with these motor symptoms of spinal cord compression, the motor symptoms of a cauda equina tumor are always those of a lower motor neuron lesion. The paralysis may be either asymmetrical or quite localized.

Ojbective sensory disturbances are rather rare early in the course of a spinal cord tumor unless, because of some mechanical disturbance, a complete transverse lesion is produced. Loss of all types of sensation may occur in a given root distribution dependent upon the location of the tumor. This is particularly true in cauda equina tumors in which the sensory loss may occur over the posterior surfaces of both thighs and about the buttocks. In most extramedullary tumors, and particularly those on the dorsal and lateral aspects of the cord, the greatest sensory loss occurs in the most peripheral dermatomes and the minimum sensory disturbance occurs *near the level of the lesion*, but *the opposite is true of intramedullary* tumors. Dissociation of sensation—preservation of tactile with loss of pain and temperature sensibility—occurs more often in intramedullary than in extramedullary tumors. The pathological process is therefore similar to that degenerative disease of the spinal cord which begins about the central canal, syringomeylia. Elsberg[1] has pointed out, however, that dissociation of sensory symptoms also occurs in extramedullary tumors. He has produced evidence from a careful study of sensation in tumor cases which tends to show that within the cord the most eccentric and most posterior sensory fibers in the spinothalamic tracts are those which are distributed to the sacral and lower lumbar dermatomes. More anteriorly and medially lie the upper lumbar and lower thoracic fibers, and still further medially and anteriorly lie the upper thoracic and cervical fibers.

Attention should also be called to the occasional sparing of the sacral segments in compression of the spinal cord from extramedullary pressure. Disturbances of vibratory and joint sense may occur whenever the entire thickness of the cord is involved, regardless of the location of the tumor, although more commonly due to pressure upon the posterior white columns. Loss of these sensibilities on one side of the body more than the other is rather certain evidence that the growth is on that side of the cord.

Disturbances of the bladder and rectum usually occur rather late in the progress of spinal cord compression by a tumor, but sooner or later they will be present. There is a considerable amount of evidence which supports the view that the fibers which innervate the bladder sphincters run in or near the gray matter of the cord, and this may explain the time of onset of these symptoms. In tumors above the lumbar cord dysuria, or difficulty in starting the urine, may be the first symptom and this may lead to retention and then a constant overflow and incontinence. Constipation is the rule in spinal cord

[1] Elsberg, C. A.: Tumors of the Spinal Cord. New York, Paul B. Hoeber Inc., 1925.

tumors and rectal incontinence is rare except following the administration of a purgative.

Fay[1] has reported several cases of spinal cord tumor in which the vasomotor line of demarcation upon the skin corresponded exactly with the upper level of the spinal cord lesion. Pilomotor reflexes may also offer some corroborative evidence of the level of compression. In far advanced cases reflex sweating may often be observed and occasionally is limited to one or two segmental areas. We have frequently induced sweating in patients by giving them hot drinks, wrapping them in blankets and injecting 1/200 grain of pilocarpine, to determine a sweating level. This has proven to be a test of considerable aid in corroborating the level of sensory loss when the latter may be somewhat doubtful. Sweating does not occur below the level of the lesion.

As in injuries of the spinal cord, tumors may produce a combination of motor and sensory symptoms due to disturbances of function of one lateral half of the spinal cord. As has been mentioned previously, this is known as the Brown-Sêquard syndrome. In cord tumors the lesion is not commonly a perfect anatomical one, and therefore the clinical symptoms may vary somewhat. It is more distinct in extramedullary tumors and is characterized by motor symptoms only on one side and pain and temperature sensory disturbances on the opposite side.

Attention has already been called to the value of the Queckenstedt test in determining the presence or absence of a spinal subarachnoid space block following a fracture-dislocation of the vertebræ. Its value in compression of the cord by a tumor is obvious. A manometric study of the effect of jugular compression, coughing, or other forced expiratory movements upon the spinal fluid pressure should be made upon every patient who is suspected of having a spinal cord tumor. Stookey[2] has reported the results of manometric studies in spinal cord tumor patients and calls attention to the value of recognizing incomplete as well as complete subarachnoid space blocks. It has been our experience that when a small tumor is present, a partial block is more commonly found than a complete block. The fluid pressure may rise rapidly but upon release of pressure on the jugulars it falls very slowly or not at all. Naffziger has called attention to the fact that many times the patient will experience pain at the level of the tumor during the Queckenstedt test, and we have

[1] Fay, T.: Vasomotor and Pilomotor Manifestations; Their Localizing Value in Tumors and Lesions of Spinal Cord; Report of 13 Verified Cases, Arch. Neurol. and Psychiat., 19, 31, 1928

[2] Stookey, B., and Klenke, D.: Study of Spinal Fluid Pressure in Differential Diagnosis of Diseases of Spinal Cord, Arch. Neurol. and Psychiat., *20*, *84*, *1928*.

also noted that phenomenon even when straining or coughing had never previously produced the pain.

As is well known, normal cerebrospinal fluid is clear and colorless. Very frequently, the fluid below a spinal cord tumor is yellow in color (xanthochromia). While this occurs in other types of spinal cord compression, it is not so common and may be looked upon as strong evidence of a new growth. Xanthochromia does not occur so frequently in extradural as it does in intradural tumors. While it is not rare in tumors of the cervical region, it is more common in tumors of the lower thoracic, lumbar cord, and cauda equina. The yellow color of the fluid may be due to dissolved hemoglobin or to the presence of cholesterin, but this is still a debatable question. The protein content of the fluid is always increased below the level of the tumor, and at the same time the cell count of the fluid remains normal. Ayer and his associates[1] have shown by double punctures that the pressure readings and protein content of the fluid differ considerably above and below a tumor. The combination of xanthochromia, increased protein content, normal cell count, and spontaneous coagulation of the fluid is known as Froin's syndrome.

Roentgen-ray evidence of the presence of a spinal cord tumor is based primarily upon the erosion of bone produced by the tumor. Meningiomas may erode the laminæ and processes of the vertebra so that one may be misled into believing that a malignant process has affected the bone. Camp[2] has called attention to the increase in size of the bony spinal canal produced by tumors and has established a normal curve of the interpedicular measurements. We have found help also in the slight changes produced in the outlines of the pedicles of the vertebræ.

In the majority of patients with spinal cord tumors, careful and repeated neurological examinations will definitely localize the tumor. However, there are instances in which a more accurate localization is desired and thereby a less extensive laminectomy is possible. Formerly, lipiodol was used as a myelographic medium but it produces definite leptomeningeal reactions, fat encystment and degenerative changes in the gray matter of the spinal cord.[3] Pantopacque became available for use during World War II and is a most satisfactory substance to use for myelography. It can be and should be removed

[1] Ayer, J. B: Spinal Subarachnoid Block as Determined by Combined Cistern and Lumbar Puncture With Special Reference to the Early Diagnosis of Cord Tumor, *Trans Am. Neurol Assn*, p 272, 1921.

[2] Camp, J. D: Significance of Osseous Changes in Roentgenographic Diagnosis of Tumors of Spinal Cord and Associated Soft Tissues, Radiology, 22, 295, 1934.

[3] Davis, Loyal, Haven, H A, and Stone, T. T Effect of Injections of Iodized Oil in the Spinal Subarachnoid Space, Jour Am Med Assn, 94, 772, 1930.

completely at the end of the procedure. If no attempt is made to remove it, it may be found within the ventricular system from which it is not absorbed. Any substance, even normal saline solution, injected into the subarachnoid space will produce a cellular reaction of varying degrees. Therefore, all of the advantages and disadvantages which accompany a myelographic study should be considered.

It may be well to point out briefly some of the common symptoms of spinal cord tumors at various vertical levels, though each patient is an individual problem in diagnosis.

Cervical Tumors of the Spinal Cord.—Above the level of the fourth cervical segment, the outstanding symptom is that of pain in the neck and back of the ear. This causes the patient to hold his head in a fixed position. Atrophy or spasm of the sterno-cleido-mastoid and trapezius muscles occur and are usually preceded by stiffness or sensations of coldness, constriction, or a feeling of suffocation.

Tumors lower in the cervical cord produce sensory and motor disturbances in the upper extremities, and reflex changes may be present before there is atrophy or paralysis. At the fifth and sixth segments, pain occurs in the shoulder or the radial surface of the forearm, thumb, and index finger. Muscular weakness involves the muscles of the shoulder girdle principally, but atrophy is most evident in the deltoid, biceps, and brachialis muscles. With tumors at the seventh and eighth cervical segments, pain occurs on the inner side of the arm and in the ring and little fingers. Muscle involvement at these lower levels is accompanied by atrophy and paralyses of the triceps, flexors of the fingers and small hand muscles.

A tumor in the lower cervical spine is illustrated very vividly by the brief recital of a young man, aged thirty-eight years, who was operated upon in 1926.

In 1918, while in an army camp, the patient noticed numbness and tingling in the fifth and ring fingers of the right hand. This came on gradually and was present for two or three years. These sensations then disappeared in the right hand but appeared in the same fingers of the left hand. He admitted rather reluctantly that for a year he had noticed some difficulty in walking, in that he had a tendency to drag his left foot.

On September 14, 1926, he consulted a doctor and a lumbar puncture was performed. The following day his left arm was very stiff, as was his neck. His walking then became impaired. At the same time, his right arm and hand again became numb. This numbness was associated with a feeling of "pins and needles" but he never had root pains.

On October 12, 1926, the left arm was spastic and paretic. He could flex and extend the forearm weakly and slowly. The same was true of flexion and extension of the fingers. The left arm could be abducted only 10 degrees. Abduction, flexion, and extension of the right upper extremity were strong. He could make a complete fist with the right hand.

The left leg was spastic and weak. The right leg was also stiff but was stronger than the left. All movements in the lower extremities were possible but weakly and poorly performed.

There was a loss of vibration sense below the clavicles. Position sense and sense of passive movements were present in the upper and lower extremities Pin-prick and temperature sensation were present everywhere but diminished. *Tactile sensation was diminished over the ulnar sides of the upper extremities* but elsewhere was normal.

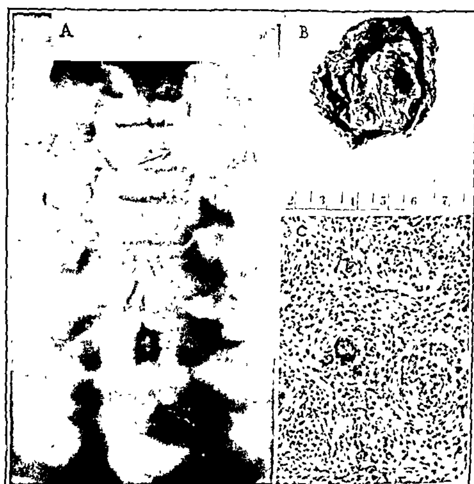


FIG 131.- Spinal cord tumor (*A*) roentgen-ray film of the cervical spine which shows erosion produced by an extradural meningeal fibroblastoma, (*B*) photograph of the gross specimen, (*C*) photomicrograph showing typical whorl formation of the tumor cells

Ankle and knee clonus were present on the left side and exaggerated deep tendon reflexes on the right. The tendon reflexes of the left upper extremity were greater than on the right The left abdominal reflexes were diminished as compared with those on the right. Bilateral Babinski reflexes were present A spinal manometric study showed a complete subarachnoid space block

Roentgen-ray films of the cervical spinal column showed a decreased density of the laminæ and transverse processes on the right side of the third and fourth vertebræ.

A laminectomy was performed upon the third, fourth, fifth, and sixth cervical vertebræ. The lamina of the fourth cervical vertebra on the right was thin and eroded. A smooth purple tumor mass beneath this lamina which extended upward beneath the lamina of the third cervical vertebra was found. It was situated laterally and pushed the cord far to the left. It was completely extradural. There was some bleeding which was controlled with muscle stamps as the tumor was removed from the attachment to the spinal canal laterally. The fifth cervical posterior root was easily dissected away from the surface.

The patient made a rapid recovery. Within two weeks, he could move the left arm above his head and could make a complete fist. The sensation of numbness had completely disappeared. The spasticity in his legs had improved so that he was able to walk without assistance.

Twenty-six years later the patient plays golf, attends to his business, and has made a complete recovery.

The tumor proved to be a meningeal fibroblastoma. (Fig. 131.)

A recital of the facts in this case emphasizes the long period which may elapse between the first symptoms of a spinal cord tumor and its ultimate diagnosis and removal. The case also illustrates the brilliant results which can be obtained following the removal of an extramedullary spinal cord tumor. Though the diagnosis could have been made when the roentgen-ray plates of the cervical vertebra were examined before operation, it was not until after the tumor had been removed that we could see very clearly the exact location and extent of the erosion.

It is extremely important in these tumors which take their origin from the meninges to remove the attachment with the tumor because if this is not done, a recurrence will take place sooner or later. One must always keep in mind the danger of dislocation of a cervical vertebra after a cervical laminectomy. This is one of the reasons why some surgeons advocate a hemilaminectomy. However, this is not a common occurrence, and the activities which these patients may obtain is illustrated in this case.

Thoracic Tumors of the Spinal Cord.—The motor and sensory segmental symptoms of the thoracic spinal cord tumors are rather scarce. Within the first two thoracic segments, however, enophthalmos, diminution of the size of the pupil, and narrowing of the palpebral fissure on the same side may occur, and occasionally vasomotor changes may be found in the face.

Paralysis of the intercostal muscles is practically impossible to detect although present investigations with action currents may afford very valuable clinical evidence in this connection. Pain in the back, girdle sensations, tenderness over spinous processes, and a difference in innervation of the abdominal recti muscles have proven of greatest benefit to us in making a localization of these tumors. They are usually mistakenly diagnosed, and this was true of a patient upon whom we operated.

In February of 1930, the patient, a female, aged fifty-six years, entered a hospital because of recurrent attacks of upper right quadrant abdominal pain. A diagnosis of cholelithiasis was made. The patient was discharged but returned in November because of continued attacks of pain with nausea and vomiting. A cholecstectomy was performed, and a gall bladder filled with stones was removed.

On March 15, 1931, the patient was admitted to the hospital because of continued right upper abdominal pain; pain in the mid-thoracic spinal column; weakness of the right leg, and anesthesia and paresthesias of the left leg.

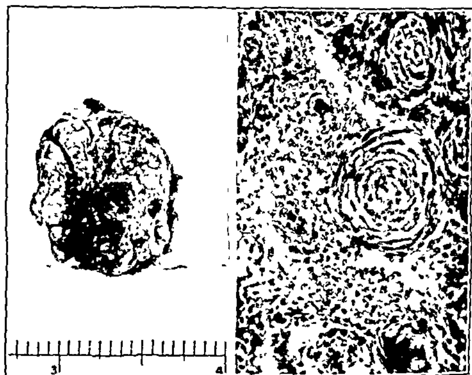


FIG. 132.—Spinal cord tumor. Photograph of gross specimen of an intradural meningeal fibroblastoma of the cervical spinal cord, and a photomicrograph which shows masses of cells arranged in whorls.

About three days after her cholecystectomy she began to complain of numbness and tingling sensations in the right leg. These symptoms increased in intensity and six weeks later were accompanied by shooting pains which ran from the foot along the lateral and posterior surfaces of the right leg to the hip. About January 1, 1931, she first noted a cold feeling in the left leg. Soon the toes of the left foot became numb.

Immediately following the operation she complained of pain in the cholecystectomy incision which was aggravated by movements of her legs. Later this pain became girdle-like on the right side in the lower thoracic area. It was spasmodic and was made worse by sneezing or coughing.

She also noted that although she urinated easily she continued to have a desire to urinate after her bladder was empty. She also complained of a lack of sensation during a bowel movement.

The upper extremities were normal. Dorsi- and plantar flexion of the right foot were weak as were movements of the toes. *Flexor and extensor movements* of the entire right lower extremity were weak as compared with the left.

There was a definite impairment of pain, temperature and tactile sensation on the left side as high as the ninth thoracic dermatome. There was slight hypalgesia and hypesthesia on the right side. Bone vibratory sense was lost in the right lower extremity.

The deep tendon reflexes of the lower extremities were exaggerated. Bilateral Babinski reflexes were present. The lower abdominal reflexes were absent, and the left upper abdominal reflex was greater than the right.

Tenderness was present on pressure over the fifth and sixth thoracic spinous process.

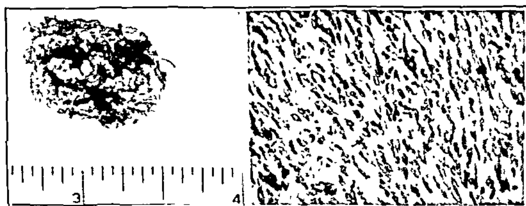


FIG. 133.—Spinal cord tumor. Gross appearance and photomicrograph of an intradural meningeal fibroblastoma removed from the thoracic spinal cord.

A partial subarachnoid space block was present.

A laminectomy was performed on the fourth, fifth, sixth, and seventh thoracic vertebræ. A reddish-purple tremor mass was found beneath the sixth thoracic spine on the posterior surface of the cord. This was intradural but outside the arachnoid. It was attached to the right side of the dural canal and a posterior root was bound down to the tumor. The root was sectioned, and the tumor removed with its dural attachment.

The patient made an excellent recovery. Her motor and sensory symptoms disappeared, and she has been completely free from pain.

Sections of the tumor proved it to be a meningeal fibroblastoma. (Fig. 133.)

It is interesting to speculate upon the character of the pain which this patient had in the upper right abdomen. There is no question whatever that a cholelithiasis was present. On the other hand, the patient was unable to differentiate between the pain which she had had before her gallbladder was removed and that which she had following its removal. At any rate, it is quite possible that in another case an abdominal operation might have been undertaken for exploration of the gallbladder without finding pathology such as was found in this case. The onset of symptoms in the extremities made it

apparent that a lesion existed outside of the biliary tract. It was later that she developed shooting pains in the right lower extremity.

Though this tumor was not particularly large, it was necessary to remove a broad attachment to the dura mater to avoid a recurrence of the growth. The attachment of a posterior root to the tumor is a sufficient explanation of the rather terrific right-sided pain which the patient experienced. The case also illustrates the fact that spinal cord tumors may grow in individuals in the later decades of life. It would have been extremely simple to have dismissed this patient as being neurotic after a cholecystectomy. The occurrence of the symptoms in her extremities prevented this misfortune.

Lumbar Tumors of the Spinal Cord.—Involvement of the lumbar segments of the spinal cord is usually accompanied by very definite root symptoms because the latter run very obliquely at that level to reach the intervertebral foraminæ from which they exit. Pain is referred to the lower part of the back and the upper part of the lower extremities; to the lower abdomen or even to the bladder and rectum. The lower abdominal reflexes remain intact, but the knee-jerk is usually diminished or abolished, and the quadriceps femoris muscle may be atrophied. While theoretically the paralyses of the lower extremities should be spastic in type, more often the extensive involvement of the roots produces a flaccid paraplegia similar to a cauda equina tumor.

A very good illustration of one of these lumbar spinal cord tumors is afforded by one of our patients from whom a chondroma of the spinal canal was removed. These are extradural tumors which arise from the intervertebral discs, grow backward, and produce pressure upon the ventral surface of the spinal cord. As the tumor is removed, it has the gross, lamellated appearance of an onion; and its tissue is elastic, tough, and stringy.

On December 7, 1930, this patient began to have an aching pain in the "small of his back" This pain left within a day or two, and he felt well until January 15, 1931. Then he began to have cramp-like pains in the muscles of the right hip This pain persisted in attacks for several days and then appeared in the right anterior thigh muscles On January 21, 1931, the same type of pain occurred in the left thigh and calf muscles.

The patient described his cramp-like pains as appearing in attacks which came on while he was shaving in the morning. They would last from ten to fifteen minutes They were severe and were increasing in frequency, but he did not become alarmed until the morning of February 12, 1931, when, as he attempted so walk to the breakfast table, he fell over against the wall and discovered that his right foot was paralyzed

The doctor advised him to go to bed and to put an electric pad upon his back This he did and received a burn over his right buttock On February 16, 1931, he noted that the left foot was paralyzed He was taken to a hospital

and several cystoscopic examinations and catheterizations were performed without finding any evidence of pathology in the genito-urinary tract. Gastro-intestinal roentgen-rays studies were performed without relief of his symptoms or solution of his difficulty.

The upper extremities were normal. When he attempted to raise either leg off the bed, there was a marked ataxia and overaction to the opposite side. Neither leg could be held extended off the bed, nor could he keep either leg in contact with the bed against resistance. Flexion of the knee against the thigh was weak on both sides, but extension of the leg was strong bilaterally. Dorsi- and plantar flexion, eversion and inversion, and movements of the toes of both feet were impossible. The muscles of the thighs and calves were soft and flaccid. There was no reaction of degeneration.

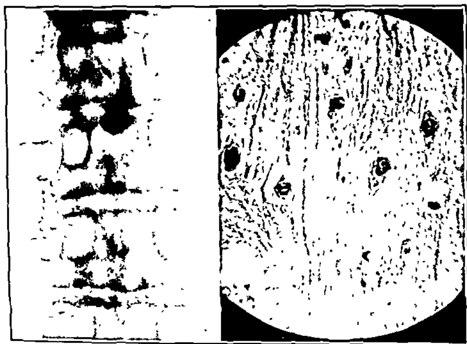


FIG. 134 —Spinal cord tumor. Roentgen-ray film which shows increased density in an intervertebral space due to an osteochondroma. Photomicrograph of the appearance of the tumor which was removed in this same case.

The knee-jerks were present but diminished The ankle-jerks were absent. Plantar stimulation elicited slight flexion of the toes. Pain and tactile sensation were diminished over the lower lumbar and sacral dermatomes. Temperature sense was recognized accurately. Vibratory sense was lost over both extremities.

A laminectomy was performed upon the twelfth thoracic and first, second, and third lumbar vertebræ After opening the dura mater, no pathology was found in the dorsal half of the dural canal. The dentate ligament on the right was severed, and the lumbar cord was rotated. Bulging into the spinal canal, anterior to the dura mater, was a yellow mass about the size of the first interphalangeal joint of the index finger. This was opposite the intervertebral disc between the first and second lumbar vertebræ. It was soft and elastic to palpation The dura was incised over this mass and immediately elastic,

whitish yellow material exuded into the canal. A large amount of this material was shredded out in lamellated layers. After it was entirely removed, the material filled a thimble.

The patient made a complete recovery with the return of all the motor power of his lower extremities and has been at his work without any symptoms since his operation.

Microscopic sections of this tumor material showed it to be chondroma.

At the time this tumor was removed, attention had not been focused so strongly upon the clinical syndrome produced by rupture of the nucleus pulposus in the intervertebral disc, which will be discussed in the following pages. However, we do not regard this as such a case although its relationship is close, as it is with similar tumors found in the cervical portion of the spinal canal.

It is difficult to find examples of spinal cord tumors limited to the lumbar portion of the cord because so many more tumors are found which involve the lower end of the spinal cord and the cauda equina. (Fig. 134.)

Lumbosacral Tumors of the Spinal Cord.—At this level we may well include the tumors of the conus terminalis of the spinal cord with those of the cauda equina.

Pain is apt to be less violent and less likely to radiate into the legs than in cauda tumors; on the other hand, sphincter disturbances are more marked; symptoms develop more rapidly and fibrillary twitchings are present in the paralyzed muscles. Pains in the small of the back, down the posterior aspects of the legs and in the perineum are usually intense in caudal tumors. The knee-jerk may be retained in conus lesions, but is almost always absent in tumors of the cauda. True incontinence or retention with overflow of urine are constant, while priapism, or involuntary ejaculation, are not uncommonly observed. The paralyses are flaccid with marked atrophy. Loss of sensation is scattered and may be quite asymmetrical, but in caudal tumors the last three sacral dermatomes practically never escape. In spite of these differences in symptomatology, as a practical matter it is very difficult to distinguish before operation between a conus and a cauda equina tumor

The long history of a cauda equina tumor is well illustrated in the record of the following case:

In 1915, when the patient was twenty years of age, she began to have burning pains about the lower part of her back. These came on in attacks which lasted several weeks after which she would be free from discomfort. She sought medical aid and was advised to have her tonsils and several teeth removed, which she did without relief. From 1915 to 1917 she was under the care of chiropractors and osteopaths, but her pain continued In 1917, it was thought possible that her pain might be due to a right inguinal hernia and a chronic appendix Therefore, a herniotomy and an appendectomy were performed She had few attacks of this burning pain in her back from that time until 1920, when the pains appeared again; and in 1921 when her first baby was born, her pains were very severe. In 1923, upon the birth of her second child, the pains became severe, sharp and shooting in both hips These

were so severe that she was placed in the hospital in a body cast for eight weeks. She obtained some slight relief from the pain and was able to do her housework In 1925, her third child was born, and her pains again became unbearable. She was again placed in a hospital, and an extension apparatus was applied to her legs for two weeks.

For five years this patient suffered with a burning, aching pain in her back and legs which changed at intervals to a sharp, shooting, excruciating pain which radiated down her legs and finally into her feet. During this time she was told that she was nervous and was given tonics. On January 25, 1931, the patient's feet and ankles became numb, and this increased, associated with hyperalgesia and paresthesias until it involved both feet, legs, and the backs of the thighs. She continued to walk with some difficulty until March 6, 1931,



Fig. 135.—Gross appearance and photomicrograph of the tumor removed from the cauda equina.

when she developed a bilateral foot-drop and a complete loss of bowel and bladder control. During all this time, coughing, sneezing, and straining produced sharp shooting pains down the backs of her thighs and her legs.

The patient could raise both thighs from the bed very weakly. The extensors of the left thigh were weaker than the right. The abductors and adductors of both thighs were weak The flexors of both legs were weaker than the extensors. Dorsi- and plantar flexion of both feet were impossible. Movements of the toes could not be performed The knee jerks were present but diminished, and the left more so than the right. The right ankle jerk was present, but the left was absent. Plantar stimulation produced a slight flexor response on the right, but none on the left.

There was a loss of sensation to pin prick over the fourth and fifth lumbar and all of the sacral dermatomes Spinal manometric studies showed a slight rise of spinal fluid pressure upon jugular compression and a very slow fall upon release.

A laminectomy was performed upon the twelfth thoracic and first three lumbar vertebræ. Below the second lumbar spinous process there was a large mass on the right side of the dural canal which was situated in front of the

conus medullaris and the roots of the cauda equina. It was yellow-gray in color and gelatinous in appearance. It was encapsulated but very soft and friable. It had enlarged the spinal canal anteriorly. It extended upward to beneath the twelfth thoracic spine and downward to beneath the third lumbar spine. As we attempted to remove it, the tumor broke; and it was necessary to remove a considerable amount of it in fragments. However, the entire capsule and contents of the tumor were removed.

The patient made an excellent recovery and is able to do her housework although her legs remain somewhat weak. She gained 28 pounds in weight, and her bladder control returned.

On microscopic section this tumor proved to be a neurofibroma and probably originated from one of the roots of the cauda equina although we were unable, because of its site, to state definitely where it originated. (Fig. 135.)

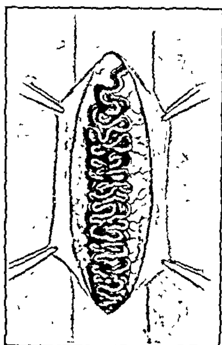


Fig. 136.—Drawing made from an operative sketch of an angioma of the cervical spinal cord.

The story of this patient over sixteen years is a grave comment upon the treatment she received only because no thought had ever been given to the possibility of a spinal cord lesion. There is no question but that this tumor grew to be an enormous size during those years, and its surgical difficulties were considerable because it completely filled the spinal canal. Moreover, its presence during fifteen years produced changes in the lower portion of the spinal cord and its roots which may be irrecoverable though the patient has been entirely relieved of pain.

Intramedullary Tumors.—Intramedullary tumors are usually gliomas although rarely other types have been recorded. Whereas extramedullary tumors bear a very close analogy to intracranial tumors, intramedullary tumors do not compare so closely. For example, astrocytomas (an adult cell type tumor) are rare in the spinal cord, whereas, they form 37 per cent of the cerebral gliomas. On the other hand, the majority of our 34 intramedullary tumors are ependymomas, a tumor which does not exceed a frequency of 4 per cent in the brain tumors.

The symptoms of intramedullary tumors are in nature similar to those of the extramedullary type. The differentiation is difficult if not impossible except on the basis of a statistical study. Many times a dissociation of sensory loss will make it almost impossible to

distinguish such a tumor from syringomyelia though the freedom of the sacral segments from sensory loss in the former is of considerable help. Most intramedullary tumors occur in the cervical segments of the spinal cord and may be well localized, though cases are recorded in which the tumor extended far down the spinal cord.

The surgical treatment of intramedullary tumors of the spinal cord is not particularly successful. The surgical procedures which allow these tumors to extrude themselves offer a better chance for less injury to the spinal cord than if removal is attempted by other means although this, of course, depends upon the course and microscopic characteristics of the tumor.

The long history and symptoms which led the patient's doctors to believe that he was suffering from a degenerative disease of the spinal cord is well illustrated in the story below:

In 1935, the patient, then forty-one years of age, fell off a load of hay and struck his lumbar spinal column. He was unable to arise immediately, but when assisted to his feet was able to walk. He dragged his right leg for several weeks following the accident.

In 1937, he began to notice that both hands and both feet were getting numb and weak. This began as tingling in the right arm and a numbness over the upper portion of the right side of the chest. Shortly afterward the numbness and tingling extended to the left arm. He was told that he had pernicious anemia and was placed on liver therapy without showing any signs of improvement. His condition became progressively worse until he became unable to walk and both lower extremities were completely paralyzed. Loss of control of the bladder and rectal sphincters occurred during the past year of his illness, and for six months he had pains in his lower extremities, which were not constant.

There was a complete spastic paraplegia present, and movements of both upper extremities were very weak. Extension of the forearms was weaker than flexion, and there was slowness and rigidity in movements of the wrist, fingers, and thumb. He was unable to form a cone with either hand or to approximate the thumbs and little fingers Fibrillation was present in the right deltoid and supraspinatus muscles Bilateral knee and ankle clonus were present, the right wrist, ulnar and biceps jerks were absent, and the triceps jerks were diminished, but the deep reflexes of the left upper extremity were increased. The abdominal and cremasteric reflexes were absent, and there was a bilateral Babinski phenomenon

There was a loss of sensation to tactile stimuli on the right side of the body from the sixth cervical dermatome downward and on the left below the first thoracic. Pin prick stimuli were not recognized below these same levels, but the loss to pain was more marked in the upper levels than below in the lumbar dermatomes. The lowermost sacral segments on the left were sensitive to tactile stimuli. Temperature sensation was lost in the same distribution.

There was a complete subarachnoid space block; the spinal fluid was yellowish in color, there was a four plus globulin reaction and a total protein content of 1800 mg. per 100 cc. of fluid.

At operation the extradural fat was missing entirely from the fourth cervical vertebra to the first thoracic and no fluid escaped when the arachnoid mem-

brane was opened. The spinal cord occupied the entire dural canal and was yellow, very vascular, and in areas appeared to be cystic. An incision was made in the median line and immediately a firm, purple mass extruded. A subtotal removal of the intramedullary growth was accomplished. Upon microscopic section the tumor proved to be an ependymoma. (Fig. 137.)

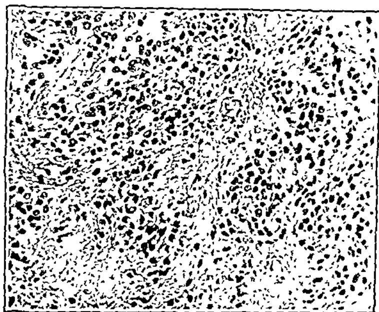


FIG 137 —Spinal cord tumor. Photomicrograph of an intramedullary ependymoma removed from the cervical spinal cord showing arrangement of cells with darkly stained nuclei in pseudo-rosette formation and a fairly abundant connective tissue stroma, × 212.

This operation was performed three years after unmistakable signs of spinal cord involvement had been present. The diagnosis of pernicious anemia was not justified on the basis of the sensory findings, but more disastrous to the patient than the wrong diagnosis was the fact that a spinal fluid examination was not done at the time a spinal cord lesion was suspected.

Syringomyelia.—*Often it is difficult to differentiate an intramedullary glioma from syringomyelia, particularly in the early stages of the latter disease, before marked atrophy of muscles has occurred*

Though it seems likely that a few sporadic cases of syringomyelia had been operated upon prior to 1926, it was Puussepp's report in that year of the results of operation upon 2 patients which focused attention upon the surgical possibilities in this disease. Puussepp's procedure consisted in exposing the distended spinal cord and incising it longitudinally along the posterior median septum; thus releasing clear fluid under pressure from within the typical intramedullary cystic cavity. Since Puussepp's paper, a large number of surgeons

have reported their individual experiences, and in each instance the immediate results, at least, have been beneficial.

Our own experience concerns 29 patients, all of whom could be considered to be in the early stages of the disease. There have been no operative deaths, and all may be said to have received some immediate benefit. One patient, operated upon sixteen years ago, was able to carry on his work as a bookkeeper, and when last examined had no detectable symptoms of progression. Another patient who had pain extending into the area of the trigeminal nerve because of the involvement of the spinal tract of the fifth nerve, has remained completely free of all pain for seven years with few motor evidences or progression of the disease. The story of one of these patients is rather typical.

The patient, aged twenty years, first noted that the grip in his right hand became weaker and his little finger was flexed. When warm weather came, these symptoms disappeared but with the onset of winter again the loss of strength became even more marked than it was originally. Then he found a loss of sensation in the right hand over the ring and little fingers and was surprised to find that while his left hand perspired, his right remained dry. Twitching of his small hand muscles and "cramps" in the right hand finally brought him to his physician four years after the original complaint.

Upon examination the right hand showed a "claw hand" deformity typical of an ulnar nerve lesion. There was marked atrophy of the hypothenar eminence and the interossei spaces. He could not form a four-fingered cone with the right hand, and he was wholly unable to adduct or abduct the fingers. The biceps and wrist-jerks were diminished, and the triceps reflex absent on the right. There was a diminution of pain and temperature sensation over the second, third, fourth, fifth, sixth seventh, eighth cervical and first thoracic segments on the right. There was definite atrophy in the trapezius and supraspinati muscles on the right. There was no spasticity in the legs and no involvement of the deep tendon reflexes of the lower extremities. A spinal manometric study showed no evidence of a partial or complete subarachnoid space block.

At operation a large distended cervical cord was exposed An incision 1.5 cm in length was made in the posterior median septum The cord was very thin and immediately clear fluid escaped under considerable pressure This was examined and found to contain no cells. The protein content was 0 200 grams per 100 cc. of fluid. The cavity was brownish-gray in color, smooth, and glistening. The cord collapsed after the release of fluid and the incision gaped open

Since his operation, his symptoms have remained stationary There has been no involvement of the left side, and the muscle atrophy has not increased. His loss of sensation has remained unchanged.

In 2 patients, both of whom had definite loss of sensation to pain and temperature stimuli in the distribution of the trigeminal nerve, this symptom has disappeared completely. In another patient pain in the upper extremity has been relieved. In still another, spasticity in the left arm and leg has been materially decreased so that the

patient is quite able to carry on his school work. In 1 patient, a second operation was performed, and the cord was incised at a higher level. There was in this case evidence of two distinct cylindrical cavities which did not communicate. We were able to show that the original incision in the spinal cord had not closed, but just how important a rôle two silver clips placed on the edges of the cord incision had played, we are unable to say. None of our patients has been made completely symptom-free, and the period over which they have been studied carefully has been too short to rule out the natural course of the disease in that particular individual.

Typical cavities of the spinal cord in syringomyelia are lined with brownish or yellowish-gray glial tissue. While small cavities may exist in the center of a large gliotic mass, we have thus far encountered only large cavities with very thin walls, which as far as gross examination could tell, were continuous with the central canal of the cord.

It is obvious that the logical criticism of this surgical procedure is that it does nothing to remedy the pathological process which underlies the disease, the etiological factor in which remains completely unknown. While this is true, there is one fact which characterizes all of the reports in the literature and which we have found to be uniformly true; namely, that the fluid released from the intramedullary cavity is under marked pressure. The release of this fluid and the reduction in intramedullary pressure is undoubtedly a factor in the results which have been noted thus far.

Deep roentgen-ray therapy has been used in the treatment of syringomyelia, and there have been many cases collected in the literature which have shown improvement. The rationale underlying such treatment is, of course, the effect of the radiation upon the glial lining of the cavity and its blood-vessels rather than upon the fluid content of the cavity.

It is very easy to obtain an erroneous impression from the cases reported in the literature, and one's own experience as to the success of myelotomy. It is probably true that an equal number of failures have remained unrecorded, and it is difficult to know how long improvement may last or whether or not the normal course of the disease in a particular patient is such that the progression of the symptoms is so gradual that the operation has played no important rôle. Perhaps combined radiation therapy and myelotomy may offer relief to these patients incapacitated by a serious disease for which no specific therapy is known.

EXTRADURAL AND VERTEBRAL TUMORS

Under this heading we may include those extradural tumors, such as the giant-cell sarcomas, Hodgkin's disease and hemangiomas; *tuberculous spondylitis, metastatic carcinoma, hypernephroma and* myelomas, all of which may produce compression of the spinal cord and its membranes, or a pathological fracture-dislocation may occur



Fig 138.—Roentgen-ray film of the cervical spinal column which shows erosion in the bone produced by a metastatic carcinoma.

in a diseased vertebra following a trifling injury which may closely resemble the symptoms and course of a fracture-dislocation from a more serious trauma.

In the majority of cases of malignant disease of the spine, the onset of symptoms of spinal cord compression is very rapid. Pains in the back, chest, or abdomen are very severe and rather irregular in distribution. *Carcinoma* of the spine is always metastatic and is quite frequent in women with carcinoma of the breast or in men with prostatic carcinoma. (Fig. 138.) A spinal metastasis from a *hypernephroma* is not rare, but the tumor increases slowly in size so that root pains may be present for a long period before spinal

cord symptoms appear. *Sarcomas* may occur primarily in the vertebra or in the soft tissues about the spine, and it is common to see an hourglass or dumbbell-shaped tumor. Sarcomas often begin about the vertebral arches so that a paravertebral swelling is commonly found. In the differentiation of malignant vertebral disease which is producing spinal cord compression, the roentgenogram offers

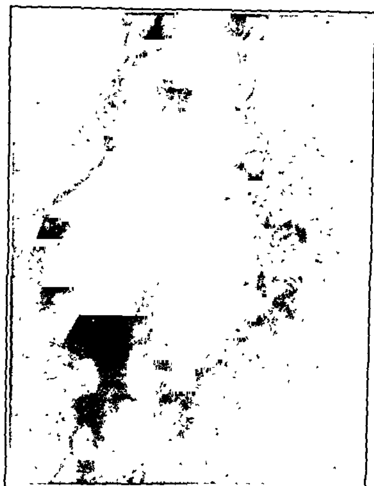


Fig 139 —Roentgen-ray film of lower thoracic and upper lumbar spinal column which shows the erosion and proliferation of bone produced by a giant cell sarcoma.

great diagnostic aid In many cases, however, the amount of destruction which exists is not actually shown Areas of erosion of the bone are characteristic, and it may be so great that the bodies of one or more vertebra may have collapsed with narrowing of the bodies and disappearance of the intervertebral discs. It has been said that primary sarcomas of the vertebræ attack the margins of the vertebral bodies and that the destruction is irregular and the intervertebral discs are not attacked primarily. It is common to find that the

compression from these malignancies of the vertebra occurs from the ventral aspect of the cord. (Fig. 139.)

Heuer[1] has given a comprehensive description of the so-called "*hourglass*" *tumors* which arise along the spinal column from the



FIG. 140 —Roentgen-ray film of thoracic spinal column which shows destruction and compression of the thoracic vertebræ produced by a tuberculous spondylitis and the production of a paravertebral abscess. The patient showed symptoms of a complete transverse lesion of the spinal cord.

highest cervical to the lower sacral vertebræ. These tumors may originate from the spinal canal (ganglions, nerves, dura, vertebral ligaments); from the vertebræ (intervertebral discs); or from outside the spine (sympathetic chain, ribs, or fascia). They have been described as growing outward through an intervertebral foramen or between two adjoining laminæ, or, arising paravertebrally, they extend through an intervertebral foramen into the spinal canal. A

[1] Heuer, G. J The So-called Hour Glass Tumors of the Spine, Arch. Surg., 18, 935, 1929.

flat endothelium. The intervascular tissue is loose and acellular; the bony trabeculæ within the hemangioma show evidences of necrosis and absorption, and strips of fibrocartilage are present from the sides of which spicules of newly formed bone project.

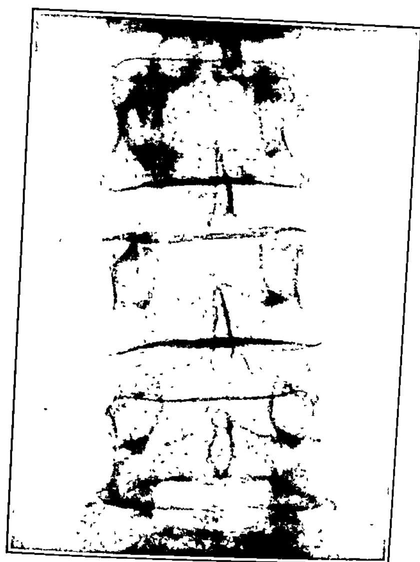


FIG 142.—Roentgen-ray film of the lumbar spine which shows an hemangioma of the third lumbar vertebra. Notice the longitudinal striations in the vertebra produced by the tumor.

Clinically, these tumors are benign, and there is no microscopic evidence of malignancy. Surgically, these lesions are difficult because of the almost uncontrollable bleeding which may be encountered. (Figs 141 and 142.)

Chordomas, a rare and usually fatal tumor which arises from the fetal notochord, may be found in the sacrococcygeal region and with continued growth produce pressure upon the spinal cord. Grossly,

these tumors are soft, jelly-like and are grayish-pink in color. They usually are very vascular and occasionally are made up of cheesy, necrotic material with small islands of viable tumor. These tumors are related to the small masses of soft jelly-like tissue often found at the autopsy table, arising from the clivus Blumenbachii (the sloping surface between the sella and foramen magnum). The presence of a painful swelling of long duration in the sacral region coupled with an irregular defect in the sacrum shown by roentgen-ray is suggestive of a chordoma. We have encountered one enormous intracranial chordoma which extended laterally from its origin so far that it produced all the signs and symptoms of a temporal lobe tumor. It was quite avascular, and its structure was that of Jello which practically disappeared when it was passed through the fixing and dehydrating agents.

HERNIATION OF THE NUCLEUS PULPOSUS

Many terms have been used to describe the clinical symptoms produced by displacement of the nucleus pulposus and the pressure which it produces upon the spinal cord or its nerve roots. A favorite term is the layman's, and some doctors', "slipped disc;" another is a "prolapsed disc;" and still another a "ruptured disc." Actually, an intervertebral disc is an oval cartilaginous structure which separates the vertebral bodies and in the center of which is the remains of the notochord, the embryological anlage of the spinal column and spinal cord. This cartilaginous disc is surrounded by a fibrous layer, the annulus fibrosus, which is weakest laterally. As a result usually of trauma which occurs most frequently following acts of flexion or torsion of the trunk while lifting heavy weights or pulling against unyielding objects, or falling, the annulus fibrosus yields and the nucleus pulposus is extruded toward the weakened point. Repeated insults may lead to rupture of the annulus fibrosus and actually extrusion of the nucleus pulposus. When this herniation, or rupture, occurs in the mid-line which is seldom in comparison with its lateral herniation, then symptoms of a spinal cord or cauda equina tumor occur. Otherwise, the symptoms are due to irritation of the overlying nerve roots at the specific intervertebral space involved.

Although it has only been within the past twenty years that herniation of the nucleus pulposus has assumed a position of prominence in the differential diagnosis of spinal and "low back" disease, observations with regard to its occurrence antedate its general clinical acceptance by many years. Virchow noted an example of protrusion of the nucleus pulposus in 1857 and emphasized the traumatic element in the clinical history. In 1911, Goldthwaite emphasized

the consequences of rupture of the nucleus pulposus through the annulus fibrosus and pointed out its effects upon the spinal nerves. In 1922, Adson operated upon two patients with protrusions of the nucleus pulposus through the annulus. In 1927, Schmorl published the first of several detailed studies on the pathological aspects of the intervertebral disc and noted nucleus pulposus herniation in 38 per cent of a large series of cadavers examined during the course of routine autopsy work. In 1928, Stookey reported a series of 7 cervical herniations of the nucleus pulposus, and in 1934 Mixter and Barr urged the intraspinal injection of radiopaque oil as a roentgenological aid in the accurate localization of nucleus pulposus herniations.

The intervertebral discs lie between the opposed surfaces of all of the movable vertebræ from the third cervical to the junction between the fifth lumbar and the first sacral vertebræ. They are thicker in the lumbar region, and their surfaces conform to the vertebral curvature. The structural elements are three: a fibrocartilaginous matrix—the annulus fibrosus—which embeds a more or less spherical mass of gelatinous, hydrophilic, mucomesenchymatous tissue of notochordal origin, the nucleus pulposus. Both of these elements are sandwiched between the third component—thin plates of hyaline cartilage which separate the enclosed contents from the spongiosa of adjoining vertebral bodies.. If one considers the hyaline tissue as representing opposed epiphyses and the semifluid nucleus to be modified joint fluid, then the annulus may be regarded as a joint capsule. In a sense, this ring of fibrous tissue serves that purpose for it attaches to the spongiosa by means of modified Sharpey's fibers, to the vertebral periosteum, and also to the anterior and posterior longitudinal ligaments. This concept is opposed by some but serves its purpose if it emphasizes the fact that the nucleus, being high in water content, possesses no inherent compressibility and transmits applied forces equally throughout the medium in which it is embedded.

The position of the annulus is maintained chiefly by its ligamentous attachments These assume importance when the structure of the posterior longitudinal ligament is considered. This band of yellow elastic tissue narrows rather sharply over the lumbar interspaces and thus affords poor support to the postero-lateral margins of the disc As was stated previously, the lumbar region, particularly the fourth lumbar interspace, is by all odds the most frequent site of disc herniation, and the protrusion is almost always in a posterolateral direction.

The fact that the lumbar discs are the largest although the most inadequately supported, that they are lowermost in position and

therefore must bear most of the weight of the vertebral column as well as a corresponding share of the flexion strain, are additional reasons why the lumbar area should be the most frequently affected.

It is obvious that "herniation" may signify either extrusion of a portion of the annular material or rupture and protrusion of the nucleus. Actually, a combination of both structures is generally found; and it seems well established that repeated subminimal trauma, as well as sudden and immediately destructive pressure, will result in displacement of the disc or its contained nuclear material. At any rate, the chief tissue changes on microscopic examination—ischemic necrosis or desiccation—are explained easily on the basis of continued pressure.

In the largest number of patients, herniation of the nucleus pulposus occurs between the L4 and L5 and L5 and S1 vertebrae. However, in a significant number of patients a herniation may occur between the cervical vertebrae and most commonly between C5 and C6 or C6 and C7. Those which occur postero-laterally produce the symptoms of nerve root irritation and those which occur posteriorly in the midline may produce symptoms of a spinal cord or cauda equina tumor. Spurling[1] has said that an accurate history, especially from an observing patient, is nearly as diagnostic as that obtained in cases of trigeminal neuralgia.

Lumbar Herniations.—Most patients give a history of onset of pain in the back, which may radiate down the course of the sciatic nerve immediately or later in the course of the disease, after a known and remembered injury, though the injury may be slight and apparently inconsequential. The posture of the patient becomes quite characteristic. He usually limps when he walks, and favors the affected extremity. He stands with a flattened lumbar curve, with his paravertebral muscles taut and board-like and usually with some scoliosis away from the side of the lesion. He prefers to stand with the heel off the floor, and with the knee slightly flexed Walking, straining, sneezing, coughing or bending usually aggravate the pain in the back and down the posterior surface of the thigh.

Tenderness may be elicited by palpation along the course of the sciatic nerve in the buttocks, thigh, popliteal space and calf. When the patient lies flat on his back and is asked to raise his lower extremity with the leg extended, there is a definite limitation of straight leg raising on the affected side. When the straight lower extremity is passively raised by the examiner until the patient complains of pain, and then the foot is passively and quickly dorsi-flexed the patient may cry out with pain.

[1] Spurling, R. G., and Grantham, E. G.: Neurological Picture of Herniations of Nucleus Pulposus in Lower Part of Lumbar Region, Arch. Surg., 40, 375, 1940.

In patients who have had repeated attacks of pain, usually diagnosed as "lumbago" or "sciatica," the buttock may hang at a lower level than the one on the opposite side. The calf muscles and even the thigh muscles may exhibit flabbiness and atrophy which can be verified by accurate circumferential measurements. One may also demonstrate weakness in motor power in the affected extremity, although this may not be reliable because the patient is invariably careful to limit his movements in those muscles which lie within the distribution of his pain.

In addition to the subjective pain, patients frequently complain of a feeling of numbness or "pins and needles" sensation in the area supplied by the sensory root which is involved. This paresthesia may be intermittent, or in chronic cases it may be a permanent loss of sensation. On testing with light pin prick or stroking with a wisp of cotton, one may frequently outline an area of sensory diminution or loss. It is common to find that the hypalgesia and hypesthesia extends beyond a single dermatome into the one above and below. There is a marked variation between individuals in the pattern of skin sensory changes, the lesion being at the same intervertebral space. The patient with a compression of the nerve roots between L5 and S1 usually complains of numbness in the lateral portion of the calf, and into the lateral surface of the heel. Another patient with similar herniation may complain of numbness on the dorsum of the foot only. We do not believe, therefore, that the sensory objective findings alone are reliable diagnostic criteria of the exact nerve roots involved.

One of the commonest of all neurological changes is the depression, or absence, of the deep tendon reflexes within the area of supply of the involved roots. The patellar reflex may be diminished with a herniation which compresses either the 4th or 5th lumbar root, and the Achilles reflex may be greatly diminished or absent when the 5th lumbar and 1st sacral roots are compressed. To elicit the patellar reflexes, the patient should be lying supine, or sitting up with the legs hanging over the side of the table. The most favorable position for careful evaluation of the Achilles reflex is with the patient prone, the knee held passively flexed by the examiner who then has complete control over the position of the leg, foot, and ankle joint.

As has been indicated, these symptoms may be intermittent. In fact, many patients who have had recurring attacks find that going to bed upon a hard mattress, the use of heat to relax the spasms in the paravertebral muscles and complete rest will relieve their symptoms so they may take up their usual activities. This is probably due to a subsiding edema of the annulus, with recurrence of symptoms when the edema returns following some new trauma. Sooner or later,

however, the annulus may rupture and the nucleus becomes extruded or the edema continues unabated and the patient suffers excruciating pain.

Most patients show a mild increase in the total protein of the spinal fluid, and presumably only the fact that most herniations occur in the lower portion of the roomy lumbar sac below the usual level for performing a spinal puncture keep these values from being substantially higher.



FIG. 143.—Herniation of the intervertebral disc: (*A*) Roentgen-ray film of the lumbar spine taken in antero-posterior position, which shows pantopacque outlining a defect in the intervertebral space between the fifth lumbar and first sacral vertebræ; (*B*) lateral roentgen-ray film showing the filling defect in the same patient.

It is our custom to insist upon the presence of a characteristic history of onset and course the typical symptoms of sensory and motor root irritation and pressure, and the unmistakable myelographic evidence obtained by the use of pantopacque solution before advising operation for the removal of the herniated nucleus pulposus. This consists simply of removing enough of the lamina of the adjacent vertebræ to identify the nerve, retract it gently and expose the protruding mass covered by a soft and weakened annulus. The nerve can be easily seen to be stretched and compressed and often, as has been said, the nucleus pulposus extrudes through a rupture in the annulus and lies free in the spinal canal. The sole purpose of the operation is to free the compressed nerve roots.

We have never combined this operation with a spinal fusion under any circumstances. Combining the operations is not to the best interest of the patient and if spinal fusion proves to be necessary in order to correct intractable low back pain because of an unstable spinal column, it had better be done as a separate operation for its specific indication. It is doubtful if congenital anomalies in the lower lumbar spine play a part in the production of the symptomatology produced by a herniated nucleus pulposus.

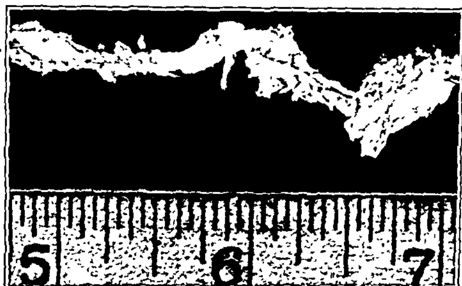


Fig 144 —Photograph of material removed from the patient whose roentgenray films are shown in Figure 143 This material extruded through the intervertebral space and compressed the roots of the cauda equina.

However, it is not enough to operate upon the patient and remove the offending nucleus pulposus These patients must be carefully and intensively rehabilitated. They should be allowed to sit up in bed on the third day post-operatively; helped to sit in a firm wooden, straight-backed chair with arms, on the fourth day and started on physical therapy on the fifth day Physical therapy should consist of heat and gentle massage to the back muscles, gradually progressing to the performance of exercises designed to strengthen their paravertebral muscles and to attain a correct posture with a lumbar lordosis without effort. These exercises, in a modified form, should be so emphasized to the patient that he continues to perform them indefinitely when he returns home and to his work. It is best to teach the patient to establish tone in his paravertebral muscles the moment he arises from bed in the morning, before he even attempts to walk across the room The patient must be advised to sit in a firm straight chair instead of a soft, overstuffed, low chair, he must keep a good

lumbar lordosis while riding in an automobile even if it is necessary to use a pillow in the lumbar curve; he must be taught to stand with his weight equally distributed on his feet and to walk erect. The use of braces, corsets, or supports of any kind to the back is prohibited because they splint the muscles instead of helping regain their tone and strength. Experiences with 954 patients in civilian practice and in a Veterans Administration Hospital have emphasized the soundness of these rehabilitation principles.

Cervical Herniations.—The earliest descriptions of herniations of the nucleus pulposus in the cervical spinal column were those of Stookey.[1] Since then Semmes and Murphey,[2] Spurling and Scoville[3] have enlarged upon the clinical syndrome by calling attention to the symptoms which result from compression of the sixth and seventh cervical roots by the herniated nucleus pulposus.

The relationship of the spinal cord to the cervical spinal canal and its fixation by the dentate ligaments, in comparison to the size of the canal in the lumbar area and the roots of the cauda equina, makes it obvious that in the former instance neurological symptoms may be more pronounced than when the cauda equina is compressed in the lumbar area by the same pathological lesion. Our own experience with this condition in the cervical region corresponds to that of all other neurological surgeons in that the herniation has occurred most frequently between the fifth and sixth or between the sixth and seventh cervical vertebræ and at both places. As has been pointed out, this is probably due to the greater strain at those points because it is the area of transition between the freely movable upper portion of the cervical spine and the comparatively fixed thoracic spine.

Pain and stiffness of the neck are usually the first symptoms and recurring attacks may occur without interval symptoms. The patient is loathe to rotate his neck, and lateral flexion toward the side of the lesion is usually more painful than flexion away from that side. The pain radiates from the shoulder, where it may be accurately localized, down the arm and into the hand. Sudden changes in the position of the head and neck, or changes in intraspinal pressure with coughing, straining, or sneezing may increase the pain and produce a sensation of an electric shock in the arm and hand. Sensations of numbness and paresthesia may be present in the hand and if the sixth cervical root

[1] Stookey, B.: "Compression of the Spinal Cord Due to Ventral Extradural Cervical Chondromas." Arch. Neurol. and Psychiat., 20, 275, 1928. "Compression of Spinal Cord and Nerve Roots by *Herniation* of the Nucleus Pulposus in the Cervical Region." Archives of Surgery, 40, 417, 1940.

[2] Semmes, R. E., and Murphey, F.: "The Syndrome of Unilateral Rupture of the Sixth Cervical Intervertebral Disc, Jour. Am. Med. Assn., 121, 1209, 1943.

[3] Spurling, R. G., and Scoville, W. B.: "Lateral Rupture of the Cervical Intervertebral Discs; A Common Cause of Shoulder and Arm Pain." Surg., Gynec. and Obst., 78, 350, 1944.

is involved the dorsal surface of the thumb and first metacarpal are particularly involved. If the seventh cervical roots are involved, the complaints are referred to the index and middle fingers. If the examiner places his hands on top of the head and suddenly exerts downward pressure, there is a sudden exacerbation of pain. The pain is aggravated by vibration or jarring, such as riding in an automobile over rough roads. Careful sensory examinations reveal a definite objective loss of sensation in the typical pattern of root distribution.

Changes in the deep tendon reflexes of the involved upper extremity are usually less pronounced than they are in lumbar herniations, and the patient usually complains of weakness or clumsiness in executing the finer hand movements. He may actually show some atrophy in the triceps and small muscles of the hand.

Roentgenological evidence is very valuable in a corroborative way because lateral films of the cervical spinal column usually show distinct narrowing of the affected intervertebral spaces and loss of the normal curve even with the neck in hyperextension. Proliferative bony arthritic changes are commonly found in other than the involved vertebræ, but narrowing of the intervertebral foramina can be demonstrated in oblique roentgenograms. We do not believe it is necessary to use pantopacque myelography to demonstrate herniations in the cervical spine. The plain roentgenograms give sufficient evidence with the physical and neurological examination. More important is the fact that there are several contraindications due to the difficulty of removing the pantopacque completely from the spinal subarachnoid space when it is placed so high. We have seen many patients, who showed residual pantopacque scattered about in the basilar cisterns where it has become encysted in the arachnoid. Often it has been seen in the ventricles and commonly along the dural sheaths of the cervical and thoracic roots where its presence may give rise to a persistent local pain.

Conservative measures consisting of traction upon the neck, bed rest, immobilization and physical therapy have been used in the 75 patients we have observed with varying degrees of success, but with recurrence of the pain. It may be necessary to employ these methods before advising surgical removal, but it must be remembered that often the radicular pain cannot be relieved and that signs of compression of the spinal cord may suddenly necessitate an immediate operation. Almost any clinical group of symptoms simulating a cervical spinal cord tumor can be produced by compression of the spinal cord by a midline herniation of the nucleus pulposus Therefore, more damage can be produced by procrastination or by failure

to recognize the possibility of this lesion than under similar circumstances in the lumbar spinal canal.

The following story of a patient is rather typical of the sequence of events and symptoms typical of this condition:



FIG. 145 —Lateral roentgenogram of cervical spinal column showing narrowing between fifth and sixth cervical vertebræ in a patient with a herniated nucleus pulposus.

A woman, aged fifty-three years, continued to be awakened from her sleep with severe pain in the right arm. The pain began in the suprascapular and supraclavicular area and radiated down the entire length of the arm to the hand. The pain was made worse by raising the arm from the side and by nodding the head to the left side; by holding the head fixed in the normal

erect position and by bending the body forward or reaching forward The pain could be decreased if she held the head forward and to the left. Numbness in the right arm along the radial side extending into the thumb and index finger had persisted from the beginning of the pain and the extremity felt colder. After she would have an "electric shock" sensation in her arm, she would suffer from a severe pruritis over the radial aspect of the right arm. She sought relief by diathermy, massage, and head traction. She had noticed no change in the strength of the right arm, but had been dropping small objects from the hand. She was known to have definite cervical arthritis for some time and had been relieved of most of the previous symptoms by living in a hot, dry climate.

There was tenderness between the spinous processes of the fifth and sixth cervical vertebræ upon direct palpation. When her head was tilted backward with the chin to the left and the occiput to the right, an "electric shock" sensation was produced in the supraclavicular region and in the radial aspect of the arm and forearm and extended down to the thumb and index finger of the right hand. There was a diminution to pin prick over the dorsal lateral surface of the right forearm, which did not extend into the hand and was not present in the arm It followed the distribution of the musculocutaneous nerve. Movements of the right hand and arm were strong and there was no muscle atrophy. The left biceps jerk was definitely more active than the right. There was distinct narrowing between the fifth and sixth cervical vertebræ (Fig. 145). There was no evidence of a subarachnoid space block and the total protein in the spinal fluid was 25 mg. per cent.

At operation a herniated nucleus pulposus was found on the right side between the fifth and sixth cervical vertebræ, which was removed. The patient made an excellent recovery and all of her symptoms have disappeared

We have had over ten times as many herniations of the nucleus pulposus in the lumbar region as compared to those which occurred in the cervical spinal column. The completeness of the recovery following operation of the latter group has been more rapid than in the cases of removal of lumbar herniations. Just as in the patient with a lumbar herniated nucleus pulposus which has been removed, so in these patients are physical therapy and rehabilitation exercises extremely important in complete and continued improvement. We have abandoned the use of cervical metal and leather braces to be worn immediately following operation Again, we believe that gradual and careful strengthening of the cervical paravertebral muscles and increasing the range of motion of the neck is more valuable than putting the muscles at rest and allowing further stiffening and weakness to occur

SPINAL ARACHNOIDITIS

There are many diseases of the spinal cord and vertebral column which may be accompanied by an inflammatory process in the spinal meninges. For example, tuberculosis of a vertebra may be accompanied by thickening of the dura and inflammatory adhesions between

the arachnoid and pia mater or the dura mater and the arachnoid. Likewise, degenerative diseases of the spinal cord, intradural tumors and osteoarthritis may be accompanied by these meningeal changes. In addition, however, there is a group of cases in which these probable etiological factors are absent but which have been termed chronic circumscribed meningitis, chronic spinal arachnoiditis, or serous spinal meningitis. The importance of this group of cases lies in the fact that they may cause symptoms of a gradually progressive lesion of the spinal cord which cannot be differentiated from the symptoms of compression due to tumors or bony pathology. Although this pathological condition had been described previously, attention was drawn to these cases by the contribution of Spiller, Musser and Martin[1] and later reports by Horsley[2] and Krause.[3]

More recently several authors have emphasized the frequency with which adhesive spinal arachnoiditis may simulate spinal cord tumors (Stookey,[4] Hassin and Andrews[5]). The adhesions of the arachnoid to the pia and dura mater may vary from fine, spider-web processes to thick, opaque bands which surround the spinal cord and produce a complete obstruction of the cerebrospinal fluid. Although true arachnoidal cysts have been described, Stookey has reviewed these case reports critically and is of the opinion that they are extremely rare and that dense adhesions of the arachnoid which have walled off the cerebrospinal fluid have been reported as cysts. These adhesions may be so extensive as to be beyond any feasible operative procedure.

Sixty-seven patients have presented various combinations of symptoms of spinal cord disease, and all have been operated upon with the verification of the diagnosis of "chronic arachnoiditis." In these patients the symptoms have been more definitely localized, and they can be more properly grouped with the cases which were originally described and which called attention to this clinical entity.

The symptoms of a transverse section of the spinal cord, a Brown-Séquard syndrome and a cauda equina lesion have all been simulated by this chronic pathological process. In no case have we ever demonstrated a complete subarachnoid space block, and this fact has furnished a definite point which we have come to rely upon in making

[1] Spiller, W. G., Musser, J. H., and Martin, E.: Cysts of the Spinal Cord, Univ. of Penna. Med. Bull., 27, 56, 1903.

[2] Horsley, V.: Chronic Spinal Meningitis, Brit. Med. Jour., i, 513, 1909.

[3] Krause, F.: Zur Kenntnis der Rückenmarkslahmungen, Verhandl. d. deutsch. Gesellsch. f. Chir., 36, 598, 1907.

[4] Stookey, B.: Adhesive Spinal Arachnoiditis Simulating Spinal Cord Tumor, Arch. Neurol. and Psychiat., 17, 151, 1927.

[5] Hassin, G. B., and Andrews, E.: Serous Spinal Meningitis (*Circumscribed*), Jour. Am. Med. Assn., 92, 877, 1929.

a differential diagnosis between spinal arachnoiditis and a tumor of the spinal cord. However, incomplete subarachnoid space blocks may be found in spinal cord tumors, and in such instances differential diagnosis is difficult. In many of our cases of arachnoiditis we have found a positive albumin reaction in the spinal fluid, but in no instance has the total protein content of the fluid been as markedly increased as is found in the presence of a tumor. Likewise, we have never found xanthochromic fluid below the level of an arachnoiditis. As a result of these clinical observations we have come to suspect the presence of an arachnoiditis when there is no subarachnoid space block or when it is incomplete if the clinical symptoms point to the diagnosis of a tumor of the spinal cord. Root pains have been characteristic of the patients in whom the lesion has been found in the cervical segments of the spinal cord, and in the patients in whom the cauda equina was involved.

The etiology of spinal arachnoiditis still remains obscure although chronic infections and trauma seem to play a leading rôle. One patient gave a history of a chronic lung abscess; 3 had definite evidence of a marked spinal osteoarthritis; 1 followed an acute subdural spinal abscess which was successfully drained; and 1 patient was an acrobat by profession whose pathology was found in the lower thoracic segments of the spinal cord. Although some have emphasized the importance of a luetic infection, in none of our patients was there a positive Wassermann reaction in the blood or spinal fluid.

Previous infectious diseases were noted in 10 of Stookey's patients, but in none of them was there a history of evidence of tuberculosis or lues. Stookey has also pointed out that with each respiratory excursion the spinal cord moves dorso-ventrally. If the cord be fixed by arachnoidal adhesions at any given point, it will move above and below Thus there is angulation with each respiratory movement. Hassin and Andrews believe that trauma and lues are the most common etiological factors. Stookey's and our own experiences speak against syphilis, particularly in the absence of serological evidence of that disease. It is not difficult to understand how any infectious disease may affect the spinal meninges and that at a later period symptoms of that involvement may develop.

The clinical course is usually a slowly progressive one which extends over a matter of several months or years. In 2 patients the onset has been rather sudden with a feeling of numbness and heaviness in the lower extremities, but the development of weakness, spasticity, and bizarre sensory symptoms has been gradual.

At operation the dura mater is not appreciably thickened nor is the extradural fat changed in amount as is so characteristic of spinal cord tumors The first evidence of arachnoiditis is obtained when

an attempt is made to incise the dura mater without opening the arachnoid, as is the general practice of neurological surgeons in operations upon the spinal cord. The thickened and fibrotic arachnoid is so adherent to the inner surface of the dura mater that it must be dissected away.. Then the opaque, thick arachnoidea is found to be adherent to the spinal cord and dense, firm trabeculæ actually suspend the cord. Often multiple pockets of thickened arachnoid contain an accumulation of fluid which must circulate even though improperly because of the absence of a demonstrable block when the Queckenstedt test is performed. These strands of fibrotic tissue may be dissected off the cord in large strips, and it is common to be able to lift the spinal cord by their attachments to it. The spinal cord grossly appears to be normal in size, color, and consistency although very often there is an apparent increase in the tortuosity and branching of the posterior spinal vessels. The thick arachnoid membrane extends outward along the spinal roots binding them to the dura mater. In the patients we have observed in whom the symptoms pointed to a lesion of the cauda equina, we found the spinal roots to be red and adherent to one another. The term "caudal neuritis" has been applied to this pathological finding, but it is without any doubt the same entity.

Elsberg and Kennedy[1] have described a non-luetic inflammatory lesion of the nerve roots of the cauda equina, characterized by sharp shooting pains in the backs of the thighs and legs. The progress of the symptoms is slow with the development of lower motor neuron symptoms and objective sensory findings which involve the sacral roots most severely. At operation, Elsberg found the roots swollen, congested, and bluish-red in color. All of the cases improved slowly after the operation.

The surgical treatment of this group of cases may be attended by brilliant results, but is indefinite in that one can never be sure that the arachnoidal adhesions are not secondary to a spinal cord degenerative disease or may exist in conjunction with a spinal cord tumor above or below the site exposed, and because the process may be so extensive as to defy complete surgical interference. Like similar intracranial pathology the complete disappearance of all symptoms alone justifies one in the assumption that no other pathology exists. Very often, liberation of the adhesions at the level of the cerebrospinal fluid block will result in a complete subsidence of symptoms. On the other hand, relief may be only temporary and later similar symptoms at a lower segmental level may develop.

The following case serves to illustrate the clinical symptoms and surgical problems which characterize this group of patients.

[1] Kennedy, F., Elsberg, C. A., and Lambert, C. I.: A Peculiar Und[illegible] Disease of the Nerves of the Cauda Equina, Am. Jour. Med. Sci., 147, 645, 19[illegible]

In April of 1929, the patient first complained that the toes of his right foot felt as if they were turned under. This sensation was disagreeable but not painful and soon involved the toes of the left foot. Two weeks after the onset his feet were tender to pressure and painful when he walked. Later his fingers became affected, and the sense of touch was less acute. He described a sensation in the skin of his palms and soles as that experienced when gasoline has touched the skin and has then evaporated, a feeling of tightness and tingling. The calves of his legs became painful and weak, and then his thighs, forearms, and arms were likewise affected.

By July 25, because of the progressive weakness of his legs, he was unable to walk and could use his hands only with the greatest difficulty.

Active movements of the upper extremities were weak and awkward. There was a marked dysmetria and ataxia which was brought out by the finger-to-nose test.

Extensor and flexor movements of both lower extremities were possible but very weakly performed. There was marked ataxia when the heel-to-knee test was performed.

Muscle and joint sense were absent in the lower extremities. Bone vibratory sense was absent as high as the crests of the ilium. Pain sensation was absent in scattered areas as high as the third thoracic dermatome. Tactile sensation was lost over both lower extremities as high as the second lumbar dermatome *and diminished as high as the third thoracic segment.*

The abdominal reflexes were absent All of the deep tendon reflexes with the exception of the left triceps jerk were absent. Plantar stimulation produced a withdrawal reflex with a crossed extensor reflex in the opposite extremity

The initial spinal fluid pressure was 40 mm. of water, and this rose only to 50 mm. after ten seconds. Upon release the pressure fell to 45 mm. The Wassermann reaction upon the spinal fluid was negative.

A laminectomy was performed on the fourth cervical to the second thoracic vertebræ Fine adhesions were present between the spinal cord and the arachnoid and between the latter and the dura mater The spinal cord was normal in color, size, and consistency. Careful exploration revealed no evidence of a tumor, for which the operation was performed.

The patient made a very rapid recovery and in December of 1929 drove his car to Arizona. In May, 1931, the patient showed no residue of his illness and has been at his work constantly without further symptoms.

Recovery from the symptoms following operation has not been so striking and complete in the spinal cases as in those of intracranial arachnoiditis. We believe this is due to the fact that, at least in our own experience, the pathology has not been so circumscribed as the original descriptions would lead one to believe Many of our patients have recovered completely from their symptoms and have returned to their normal positions in life The remaining cases were definitely improved as has been proven by careful follow-up examinations, but complete restoration to normal function cannot be claimed. Two of these latter patients have succumbed three and four years after operation, but the opportunity has never been presented to make a careful pathological study of the degenerative changes which may have occurred within the spinal cord itself.

CHAPTER IX

INJURIES OF THE PERIPHERAL NERVES

"Nerve wounds are still a mysterious and disturbing problem to many doctors."—J. DEJERINE.

THE important contribution to injuries of the peripheral nerves have come as the product of three wars. The most important and earliest contribution to this subject came from Weir Mitchell's study of soldiers injured during the Civil War. From World War I peripheral nerve studies, came valuable diagnostic data but the results of operation could not be evaluated because of poor postoperative records and follow-up examinations. Although there was considerable promise that the patients operated upon in World War II would provide excellent data for study, this has not proven to be true and again the reason lies in the very poor records which were kept at the time of operation and postoperatively. Five study center groups were granted financial aid by the Veterans Administration for a follow-up study of the treatment of peripheral nerve injuries suffered in World War II. The lack of an accurate record of what was done at operation has kept these examinations from having significant value.

We have had the opportunity of examining the results of operation in 985 peripheral nerve injuries which occurred in World War I;[1] 258 patient in civilian practice, and 300 patients examined thoroughly who were operated upon during World War II.

The statistics compiled from World War I wounds showed that the radial was the nerve most frequently injured, but in civilian practice in our series, the ulnar was by far the most frequently involved. Actually, there were 88 ulnar nerve lesions, 40 lesions of the brachial plexus, 34 median nerve injuries, 28 radial nerve injuries, 26 combined median-ulnar nerve lesions, 18 peroneal injuries, 15 injuries of the sciatic nerve and 8 tibial nerve lesions.

Lacerated wounds of the upper extremity are by far the most common causes of injuries to the peripheral nerves, and the rapid increase in the incidence of automobile accidents has produced a proportionate rise in the frequency. In addition, however, we have noted many other etiological factors, some of which are peculiarly characteristic of particular peripheral nerves. For example, the

[1] Pollock and Davis have published an extensive study of 985 peripheral nerve injuries observed during World War I, cf. Peripheral Nerve Injuries, New York, Paul B. Hoeber, Inc., 1933.

radial nerve is particularly susceptible to injury in civil life because of its length and spiral course about the humerus Fracture of the humerus, with subsequent compression by a cast, splint, or callus formation; dislocations of the shoulder; direct injury during operations upon the upper extremity; malposition of the arm during anesthesia; and sudden stretching have all been observed as etiological factors.

Wounds from knife stabs, glass splinters; suicidal attempts by cutting the wrist; injuries, fractures, and dislocations about the elbow; and compression by a tourniquet, splint or cast are the most common causes of ulnar and median nerve paralysis. In addition to gunshot and stab wounds, *injury to the sciatic nerve may result* from fracture of the femur, and pelvic bones. In one of our patients it was injured by a deep hypodermic injection introduced into the buttocks.

The level of the lesion in the upper extremity, as might be expected, was in the majority of instances at the wrist. The question immediately arises as to the nature of the etiological agents which would produce isolated nerve injuries at that level. This apparent improbability is easily explained by the fact that the largest number of patients also had a division of one or more of their flexor tendons, which had been sutured by the doctor who first saw the patient after his injury. In contrast to the very creditable tendon operation, *the nerve injury had been entirely overlooked. In not a few patients* the distal end of the flexor palmaris longus had been sutured to the central end of the median nerve, or *vice versa*, under the impression that a perfect union of a divided nerve had been accomplished. These experiences of others have emphasized to us the necessity of choosing a few simple and practical methods of examination which *would allow us to determine the presence or absence of a nerve lesion* in a freshly acquired wound.

DIAGNOSIS

Perhaps the most important fact to be remembered is that a patient with a wound at the wrist which divides the median and ulnar nerves may flex his hand and fingers quite adequately. This is not difficult to understand because the flexors of the wrist and fingers receive their innervation rather high in the forearm. Therefore, to ask the patient to make a fist as a test for a nerve injury may lead to an erroneous conclusion. The most accurate tests for injury to the median and ulnar nerves at the wrist are those which involve the actions of the small hand muscles. Though several may be employed, if the patient is asked to oppose the ball of the

thumb to the tip of the little finger without flexion of the distal phalanx of the thumb or little finger, a perfectly adequate and accurate test for median nerve function will have been employed. Similarly, function of the ulnar nerve distal to a lesion at the wrist may be tested by having the patient abduct and adduct the extended fingers. If the site of injury be in the arm, to these tests may be added the request that the patient scratch the top of the table with the nail of the index or little finger while his hand remains flat. These are simple and accurate tests which can be relied upon because these movements cannot be exactly imitated, even in older injuries, by supplementary movements.[1]

In addition to these tests for motor function, one may also rely upon a loss of sensation if just a few simple facts are borne in mind. Unlike tests of motor function these apply equally well whether the level of the injury is at the wrist or in the arm. In a median nerve lesion, for example, the area of loss of sensation to pin prick which is unaffected by an overlap of function from the radial and ulnar nerves is the dorsal and palmar surfaces of the distal phalanx of the index and middle fingers. This is the isolated sensory supply to pin prick of the median nerve. The dorsal and palmar surfaces of the fifth finger constitute the area of isolated supply to pin prick of the ulnar nerve, and in a like manner the isolated supply to pin prick of other peripheral nerves has been determined. The importance of these facts is, first, in their disagreement with what has been taught to be the anatomical supply of the nerves and, second, a response to a pin prick stimulus in the anatomical distribution may be interpreted wrongly as a sign of return of function or an incomplete lesion of the nerve. Although this overlap of sensory function by uninjured adjacent nerves would not apply so dogmatically to the patient with a recent injury, it is an important factor in older injuries, and one may avoid errors by adopting the same plan of examination for both types of cases.

Pollock[2] has called attention to the overlap of pain and touch sensation in peripheral nerve injuries These facts, which were brought out by his careful analysis of a large number of cases observed during World War I have an important bearing upon the diagnosis of peripheral nerve lesions and the signs of recovery of function.

[1] Pollock, Coleman, and others have emphasized the necessity of recognizing supplementary muscle movements. In many instances the movement performed by a particular paralyzed muscle may be simulated so closely by adjacent unparalyzed muscles as to defy detection except by the most careful examination. *Cf.* Pollock, L. J.: Supplementary Muscle Movements in Peripheral Nerve Lesions, Arch. Neurol. and Psychiat., 2, 518, 1919, and Coleman, C. C.: The Interpretation of Muscle Function in Its Relation to Injuries of the Peripheral Nerves, Surg., Gynec. and Obst., 31, 246, 1920.

[2] Pollock, L. J.: The Pattern of Sensory Recovery in Peripheral Nerve Lesions, Surg., Gynec. and Obst., 49, 160, 1929.

Though the intentions are of the best, we may arrive at the wrong conclusion by testing for loss of sensation by inaccurate methods. It is quite simple to drop a large-headed pin into the barrel of a glass syringe, allow the point to extend through the tip and then replace the plunger. The weight of the latter insures an accurate and unvaried degree of pressure when the pin pricks the skin with the syringe held by the barrel. A wisp of cotton is also a simple but accurate method for testing tactile sensibility; whereas, touching the skin with a pencil or the finger stimulates other sensations.

To the failure to use a simple method of examination for loss of nerve function may be added the rather prevalent feeling that divided nerve ends will seek each other and grow together again without the surgeon's aid. These two factors alone, we believe, have accounted for the fact that 90 per cent of our cases were referred to us many months after the original injury when the deformities produced by muscle atrophy, trophic ulcers, ankylosed joints, and contractures were added to the clinical picture.

Radial Nerve.—The deformities of the hand which follow lesions of the three major peripheral nerves in the upper extremity are quite characteristic, and in themselves point to the correct diagnosis. The main deformity of a radial nerve lesion is a "wrist-drop." It is necessary only to attempt to perform movements of the fingers and the thumb with the wrist in full flexion to realize how disabling this deformity may be The patient with a radial nerve lesion cannot completely make a fist or grasp objects in his hand because he cannot completely flex the fingers when the wrist is in flexion.[1] Many times the thumb interferes with flexion of the index finger, and the patient moves it aside with the unaffected hand. These patients find it very difficult to abduct and adduct the fingers unless the hand is placed flatly upon the table, though the interossei muscles are innervated by the ulnar nerve. In none of our patients was there a complete loss of extension of the forearm. The long head of the triceps muscle receives its supply immediately after the radial nerve emerges from beneath the teres major tendon, and in none of our cases was the site of injury at that high level. Fracture of the humerus was the most common etiological agent, and the nerve was injured at the time of the fracture or was involved later by the formation of callus In 3 patients, the radial nerve was injured by a stab wound on the lateral aspect of the middle third of the arm as the nerve passes spirally about the humerus (Fig. 146.)

[1] This deformity is a striking example of the efficiency of the hand when it is in the position of normal function, to which attention has been drawn many times It also emphasizes the fact that maintaining the position of function should be the main object of any splints which may be applied to correct a deformity of the hand

Weakness of the wrist extensors may be observed when the patient attempts to raise his hand against the action of gravity. In one of our patients, whose injury to the radial nerve was below the elbow, the extensors of the fingers were paralyzed without an involvement of the wrist extensors. In another patient in whom radial nerve paralysis followed after he had used an axe to chop down a tree, a complete recovery occurred without surgical interference except in the extensor of the middle finger. We have also observed paralysis of the extensors of the wrist without involvement of the fingers, as well as the opposite condition.

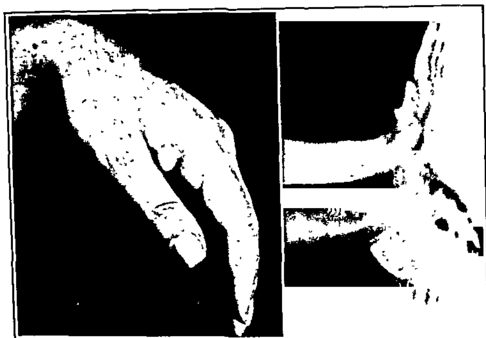


FIG 146 —Wrist-drop deformity of a radial nerve lesion, inability to dorsi-flex hand as compared to normal hand.

In judging the extent of muscle involvement following a radial nerve lesion, the development of supplementary muscle movements to which we have referred above must be kept constantly in mind. For example, in some patients strong contraction of the pronator radii teres muscle will produce extension of the wrist. This trick movement can be differentiated, however, if one carefully observes that the hand deviates to the ulnar side. Again, to demonstrate that the first phalanges of the fingers are immobile it is necessary to have the wrist passively semi-extended. Otherwise the patient may contract the interossei muscles and thus extend the terminal phalanges which in turn makes it possible for him to tense the

proximal phalanges. In one instance we have seen extension of the first phalanx of the index finger produced by strong adduction and apposition of the thumb against the first phalanx of the index finger which was, as a result, passively lifted dorsally. Though never as complete or as strong as when the extensor pollicis longus muscle is spared, the abductor and adductor pollicis and the flexor pollicis may extend the distal phalanx of the thumb. All of these supplementary muscle movements must be examined carefully to form an accurate conclusion not only as to the degree of paralysis but in order to determine evidence of motor recovery.

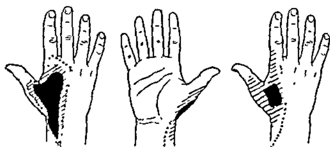


FIG 147.—Types of loss of sensation in a radial nerve lesion.

The loss of sensation in a radial nerve lesion is often so small that it may be overlooked very easily, but without a great deal of harm to the patient. The isolated supply of pin prick sensation lies between the metacarpals of the thumb and index finger on the dorsum of the hand. This small and variable loss of sensation in a radial nerve lesion is explained by the fact that the median, musculocutaneous, and ulnar nerves all take part in the sensory supply of the dorsum of the hand. (Fig 147.)

Vasomotor or secretory disturbances, in our experience, are so rare in uncomplicated radial nerve injuries, in comparison with median and ulnar nerve lesions that they do not characterize the common clinical picture. On the other hand, three or four months after injury, muscle atrophy on the dorsum of the forearm may become so marked that it appears as if the skin and subcutaneous tissue were tightly stretched over the radius and ulna.

Median Nerve.—The paralysis and marked atrophy of the small hand muscles which follow a median nerve lesion produce a striking and characteristic clinical picture. The thenar eminence becomes flattened, and the metacarpophalangeal joint of the thumb stands out like a bony knob over which the skin is drawn tightly. The thumb falls back to be in the plane of the palm, and as a result the typical "ape-hand" is seen.

Practically all of the median nerve lesions in our series occurred at the wrist as the result of severe lacerations from automobile accidents and knife wounds. In one case, a porcelain water faucet handle broke, and the patient sustained a laceration on the thenar side of the palm of the hand, which severed the cutaneous branches of the median to the index finger and thumb. While all motor func-

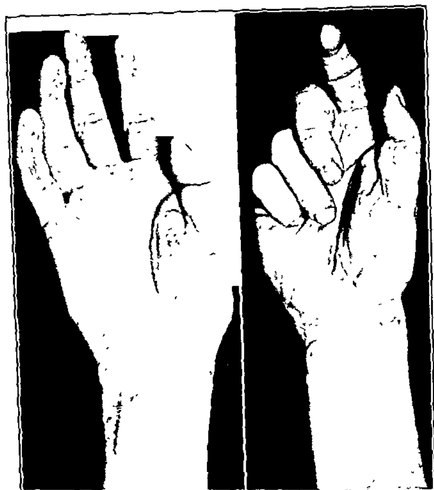


Fig. 148 —Typical deformity in median nerve lesion Note the flattening of the thenar eminence and the tendency of the thumb to fall back into the plane of the palm.

tions were perfectly retained, the patient had more than an ordinary disability from his sensory loss since he was a dentist by profession. Stab and lacerated wounds in the arm frequently injure the median nerve, and in one of our patients the healed scar on the medial aspect of the arm was so insignificant in appearance that it was difficult to regard it as the original lesion. Again fractures about the elbow joint and the lower end of the radius may often cause a paralysis of the median.

Paralysis of the flexor sublimis and the lateral portion of the flexor profundus digitorum muscles, due to a median nerve lesion in the arm, is evidenced by an absence of flexion of the index finger and feeble flexion of the middle finger. We have seen the latter finger influenced by movement in the ring finger, and occasionally the



FIG. 149 — Note inability of patient with median nerve lesion to make complete fist as compared to normal hand

deep flexor may be supplied by the ulnar nerve. When the patient attempts to make a fist, the index and middle fingers flex only slightly. Paralysis of the flexor longus pollicis makes it impossible for the patient to flex the distal phalanx of the thumb. Apposition and abduction of the thumb at right angles to the palm are impossible because of paralysis of the thenar muscles.

It would seem impossible to mistake a median nerve lesion in the presence of these marked losses of motor function In old cases, however, supplementary movements may have been acquired by the patient to such a degree that the closest examination leaves

one in doubt. For example, apposition of the thumb to the little finger may be simulated by the action of the adductor pollicis combined with the inner head of the flexor brevis pollicis and flexion of the terminal phalanges of the little finger. The one movement which cannot be imitated is flexion of the terminal phalanx of the index finger.[1] (Figs. 148 and 150.)

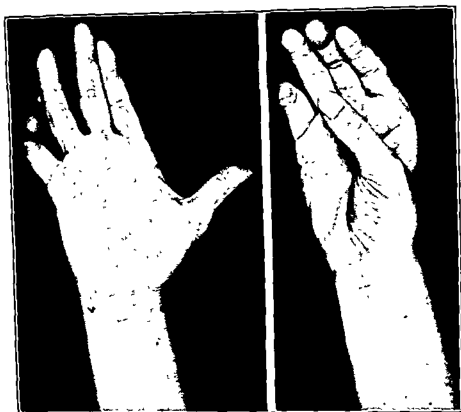


Fig. 150.—Inability of a patient with a median nerve lesion to appose the thumb and little finger.

[1] Wartenberg has described a test for median nerve function which emphasizes the importance of the median thenar muscles as the primary abductors of the thumb The ulnar nerve does not participate in abduction of the thumb and although the radial nerve may, it rarely suffers injury at the same time as the median. Wartenberg's test is equally applicable for lesions of the median nerve at the wrist or higher in the upper extremity. The patient is asked to stretch the thumbs of both hands as much as possible but at the same time they must be kept on a plane with the palm while the other fingers remain adducted. The hands are then lifted with the palms facing the examiner. In this position, the radial tips of the index fingers touch each other at a horizontal level and so do the tips of both thumbs if there is no lesion of the median nerve. However, if a median nerve lesion exists, the tip of [illegible]
the other [illegible]
proximal t [illegible]
Gynec. and Obst., 73, 872-873, 1941.

Examination of the area of isolated supply to pin prick of the median nerve has already been mentioned as an accurate diagnostic method. We have seen a loss of sensation to pin prick varying from the area of isolated supply to the accepted anatomical supply of the median. The important thing to remember is that an area of sensory loss less than the anatomical distribution may not be accepted as evidence of a partial lesion nor as a sign of recovery of function after operation. The large overlap of the radial nerve to the palm of the hand accounts in a large measure for the variation in size of the area of sensory loss. (Fig. 151.)

The skin of the palm in median nerve lesions often is purplish, cyanosed, red and cold, and it may be dry, chapped, and at times

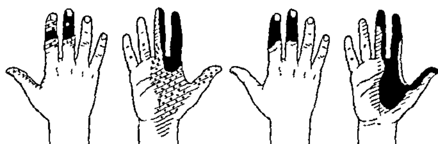


FIG 151 —Types of loss of sensation in a median nerve lesion

scaly. Ridging and hypertrophy of the nails occur, and in contrast with the normal hand the nails are pinched-in at their sides so that the surface becomes more convex So-called trophic ulcers occur commonly on the distal phalanx of the index finger as a result of cigarette burns. (Fig. 152.)

A rather large number of median nerve lesions, and not infrequently sciatic nerve injuries, may be characterized by the predominance of pain. This type of lesion was described first during the American Civil War by S. Weir Mitchell, who characterized its intensity under the name of *causalgia*. In such cases total paralysis of the muscles below the level of the wound is rare, but some weakness in movement is always present in the flexors of the index finger and in the thenar muscles

Nothing has been added to the description which S. Weir Mitchell gave of this clinical entity known as *causalgia*. His description is so comprehensive that it may be quoted extensively because of its clinical value and historical interest.

"The seat of burning pain is very various; but it never attacks the trunk, rarely the arm or thigh, and not often the forearm or leg Its favorite site is the foot or hand In these parts it is to be found most often where the nutritive skin changes are met with, that is to say, on the palm of the hand, or palmar part of the fingers, and on the dorsum of the foot, scarcely ever on the sole of

the foot or the back of the hand. Where it first existed in the whole foot or hand, it always remained last in the parts above referred to, as its favorite seats.

"The great mass of sufferers described this pain as superficial, but others said it was also in the joints and deep in the palm. If it lasted long, it was referred to the skin alone.

"Its intensity varies from the most trivial burning to a state of torture, which can hardly be credited but which reacts on the whole economy, until the general health is seriously affected.



FIG 152.—Sweating test with a median nerve lesion as compared to normal hand.

"The part itself is not alone subject to an intense burning sensation, but becomes exquisitely hyperesthetic so that a touch or a tap of the finger increases the pain. Exposure to the air is avoided by the patient with a care which seems absurd, and most of the bad cases keep the hand constantly wet, finding relief in the moisture rather than in the coolness of the application.

"As the pain increases, the general sympathy becomes more marked The temper changes and grows irritable, the face becomes anxious and has a look of weariness and suffering. The sleep is restless and the constitutional condition reacting on the wounded limb exasperates the hyperesthetic state so that the rattling of a newspaper, a breath of air, another's step across the ward, the vibrations caused by a military band, or the shock of the feet in walking give rise to increase of pain. At last the patient grows hysterical, if we may use the only term which covers the facts He walks carefully, carries the limb tenderly with the sound hand, is tremulous, nervous, and has all kinds of expedients for lessening his pain

"The skin affected in these cases was deep red or mottled, or red and pale in patches. The epithelium appeared to have been partially lost, so that the cutis was exposed in places. The subcutaneous tissues were nearly always shrunken, and when the palm alone was attacked, the part so diseased seemed to be a little depressed and firmer, and less elastic than common. In the fingers there were often cracks in the altered skin, and the integuments presented the appearance of being tightly drawn over the subjacent tissues. The surface of all the affected part was glossy, and shining as though it had been skillfully varnished."

The pathology and pathogenesis of causalgia are unknown. In fact, there is some confusion as to what constitutes this condition. Some believe that causalgia, Sudeck's atrophy, painful osteoporosis, and other so-called "minor causalgias," or painful "phantom limb," are different clinical manifestations of the same underlying disorder. The similarity, however, is remote and it is our opinion that causalgia varies sufficiently in severity to be considered as a clinical entity.

Mayfield and Devine[1] have presented data from 15 cases which were studied to correlate subjective and trophic changes with blood flow in the part. The results of their studies suggest that the pain of causalgia does not result from alteration in the blood flow, because some patients were found in whom the injured extremity was in a state of vasodilatation, while others showed vasoconstriction. The trophic changes and the response to heat and cold varied considerably with the blood flow and the severity of the trophic changes increased with prolongation of the symptoms.

Twelve patients of this group were relieved by sympathectomy of the involved limb, 1 was cured by artificial fever, and 2 recovered spontaneously. Neurolysis upon the injured nerve and periarterial sympathectomy at the level of the injury were not of benefit. Procaine block of the sympathetic chain afforded complete relief temporarily but no case received lasting benefit from repetition of this procedure. Preganglionic sympathectomy which included the site of the nerve injury gave immediate and lasting relief in 12 patients and it is Mayfield's and Devine's opinion that it should be done early to prevent stiffness of the joints due to disuse. The personality

[1] Mayfield, Frank H., and Devine, John W.: "Causalgia Surg., Gynec and Obst., 80, 631, 1945.

changes always present during the painful stages of this condition are secondary to the pain and there was no evidence in this group of cases that there was a predisposing constitutional psychic factor in causalgia.

Ulnar Nerve.—Like injuries of the median nerve, ulnar paralysis produces a characteristic deformity of the hand which is very strik-



FIG. 153.—Note the clawed hand defect which follows an ulnar nerve lesion.

ing. When the lesion to the nerve is above the wrist, these patients are unable to flex the proximal or distal phalanges of the ring and little fingers; to abduct or adduct the fingers when they are extended; to adduct the thumb; to contract the flexor carpi ulnaris; and to abduct or appose the little finger. As we have said before, when the lesion is at the wrist the flexot carpi ulnaris and the flexor muscles of the little and ring fingers are spared. The most striking symptom

to our mind is the atrophy of the small hand muscles which occurs rapidly and from which recovery is very slow and often doubtful. The atrophy in the first dorsal interosseous space is very marked and that present in the other interosseous spaces gives to the hand a skeleton-like appearance. The "clawed-hand" deformity results from the unopposed action of the extensor communis digitorum. We have seen this limited to the little finger alone, but more often

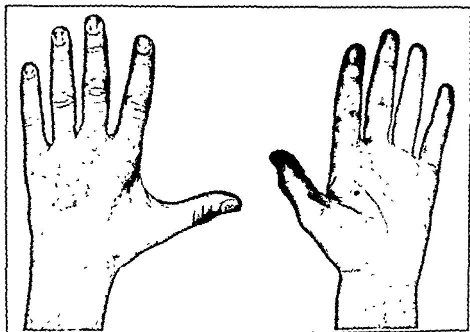


Fig 154 —Note marked atrophy of hypothenar eminence and interosseus spaces in ulnar nerve lesion

the ring finger is also involved, and occasionally the middle finger is clawed as well. The extensor communis draws these fingers into extension at the metacarpophalangeal joints, and as a result the distal phalanges become flexed The hypothenar eminence may become so atrophied that only a thin band of tissue may be palpated. (Fig. 154.)

A few simple tests will bring out the great loss of function which follows this muscle paralysis. The patient cannot appose the little finger, nor can he form a cone with the fingers and the thumb. When the fist is closed, the ring and little fingers may be imperfectly flexed The patient is unable to grasp any object, such as a folded newspaper, between his thumb and index finger. When he is asked to hold it tightly, he flexes the second phalanx of the thumb vigorously and presses the tip awakwardly against the outer margin of the first phalanx of the index finger.[1] (Figs 155, 156 and 157.)

[1] This test was first described by Froment and is known as the newspaper sign

Just as is the case in radial and median nerve paralyses, the patient with an ulnar palsy may develop supplementary movements which lead to confusion in determining the exact extent of motor loss. For example, the extensor pollicis longus muscle is the prime mover

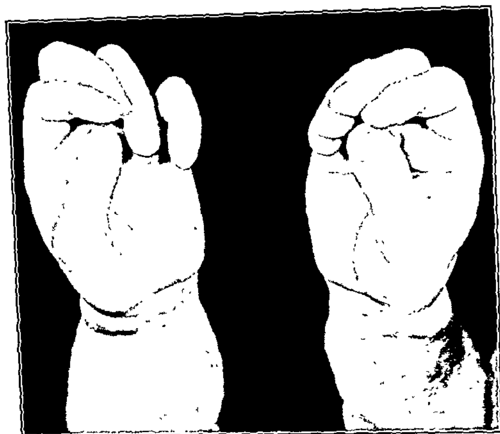


Fig. 155.—Inability of patient with an ulnar nerve lesion to form a cone with the fingers.

in adduction of the thumb, and in ulnar nerve lesions it may wholly supplant the loss of the adductor muscle of the thumb. Even abduction of the fingers away from the midline may be accomplished by forcibly extending the first phalanges, particularly of the index and little fingers.

The most extensive loss of sensation in an ulnar nerve palsy occupies the ulnar border of the palm, the palmar and dorsal surfaces of the little and the ulnar half of the ring fingers. This is readily recognized as the anatomical distribution of the nerve. However, the isolated area of loss to pin prick sensibility may occupy only the palmar and dorsal surfaces of the little finger and a small triangular area on the dorsal surface of the hand over the fifth metacarpal bone. Again we wish to emphasize that the rest of the area



FIG. 156.—Inability of patient with an ulnar nerve lesion to grasp a newspaper between the thumb and index finger without a vigorous flexion of the second phalanx of the thumb.



FIG. 157.—Note inability of patient with ulnar nerve lesion to abduct and adduct fingers and thumb.

anatomical distribution may be sensitive because of overlap from the median and radial nerves The use of this very practical point in drawing conclusions from sensory loss has proven a great help in our hands. (Fig 158)

As is true in median nerve lesions, trophic and vasomotor changes are well marked in ulnar nerve lesions. The little finger is usually cold, dry, and a purplish discoloration is present The little finger nail is ridged, hypertrophied, and deformed. In our group of patients, flicking cigarette ashes away was the most common cause for the ulcerations which were often present at the tip of the little finger

Not infrequently ulnar palsy develops many years after the original injury which may not be associated with the symptoms in the hand by the patient One of our patients in this group of *tardy ulnar*

paralyses had received a fracture about the left elbow twenty years previously as a boy of ten. Gradually a loss of sensation and motor function appeared until he was greatly handicapped. At operation the ulnar groove was practically unrecognizable, and the nerve was

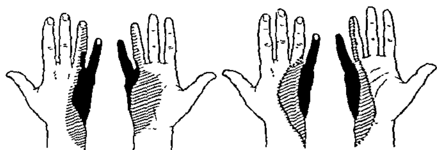


FIG. 158.—Types of sensory loss in ulnar nerve lesions.



FIG. 159.—Note the marked atrophy of the thenar and hypothenar eminences in a patient with a combined median-ulnar nerve lesion. This produces the typical ape hand.

riding over a bony callus each time the elbow joint was moved. When he was examined four years after the nerve had been transposed to the anterior surface of the elbow, all sensation had returned to normal; the thin thread of muscle fibers between the thumb and first metacarpal had been restored to a mass measuring 1½ inches in

thickness, and all of the small hand muscles supplied by the ulnar performed their normal movements.

Combined Median-Ulnar Lesion.—Only a few of our 26 patients with combined median and ulnar paralysis resulted from lacerated wounds in the arm. The majority were the result of lacerations at the wrist. The rest, with one exception—a fracture about the elbow joint—were the result of lacerations at the wrist. All of this latter group originally had severed flexor tendons but came to us after the tendons had been repaired, and evidences of median and ulnar



FIG. 160.—Sensory loss in a combined median-ulnar nerve lesion.

nerve involvement were far advanced. They illustrate so vividly the patience and painstaking care which must be exercised, and these under proper surgical conditions, to be sure that all of the divided structures are repaired following a severe laceration of the wrist.

The deformity which follows a complete lesion of these nerves resembles an ape's hand even more closely than does median nerve paralysis. The wrist is slightly hyperextended, and the hand inclines to the radial side The thumb is abducted and lies in the palm of the hand while the first phalanges are extended moderately and slight passive flexion of the last two phalanges is present. Muscular atrophy is marked in the hypothenar and thenar eminences and in the dorsal interosseous spaces (Fig. 159.)

Sensation to light touch is lost over the palmar and dorsal surfaces of all the fingers, excepting the area supplied by the radial nerve and the ulnar border of the dorsum of the hand. In our cases the loss of sensation to pin prick varied considerably according to the degree of overlap of function from the radial and musculocutaneous nerves. In one patient the thumb, radial side of the palm, proximal phalanges of the index, middle, and part of the proximal phalanx of the ring finger were sensitive (Fig. 160.)

Vasomotor, secretory, and trophic changes which we have seen in these combined injuries are similar to those found in cases of paralysis

of the ulnar and median nerves alone. However, in one patient, who had been shot in the arm, there was an injury to the brachial artery, and the skin was cold, cyanotic, glossy, and edematous while the nails were hypertrophied and deeply ridged.



FIG. 161.—Sweating test in patient with a combined median-ulnar nerve lesion.

Brachial Plexus.—Traumatic lesions which involve parts of the brachial plexus are not uncommon in civilian and military life. They may be the result of penetrating stab or bullet wounds, or more commonly in civil life, are produced by blunt trauma upon the side of the neck with the head flexed far to the opposite side and the shoulder depressed. Birth palsies result from injury to the brachial plexus or its roots during parturition, especially in breech deliveries, and the mechanism of the trauma is the same. The chief factor is over-stretching and traction upon one or more components of the plexus produced by any means which force the head

Nerves	*Muscles*	
Phrenic (C.3,4,5)	Diaphragm	
Long thoracic (C.5,6,7)	Serratus anterior	
Thoracodorsal (C.5,6,7)	Latissimus dorsi	
Upper subscapular (C.5,6)	Subscapularis	
Lower subscapular (C 5,6)	Subscapularis Teres major	
Suprascapular (C.5,6)	Supraspinatus Intraspinatus	W
Lat. ant. thoracic (C.5,6,7)	*Pectoralis major*	
Med ant. thoracic (C.8, T.1)	Pectoralis minor	
Axillary (C.5,6)	Deltoid Teres minor	P
Musculo-cutaneous (C.5,6,7)	Coracobrachialis Biceps brachii Brachialis	
Median (C.6,7,8, T.1)	Pronator teres Pronator quadratus Flexor carpi radialis [illegible] Opponens pollicis Flexor pollicis brevis, lateral ½ Lumbricales, 1 2	
Radial (C 5,6,7,8, T.1)	Triceps Anconeus *Brachioradialis* Extensor carpi radialis longus Extensor carpi radialis brevis Extensor digitorum communis Extensor digiti quinti proprius Extensor carpi ulnaris Supinator Abductor pollicis longus Extensor pollicis longus Extensor pollicis brevis Extensor indicis proprius	W (Triceps, Anconeus)
Ulnar (C 8, T.1)	Flexor carpi ulnaris [illegible] Interossei Lumbricales, 3 4 *Adductores pollicis* Flexor pollicis brevis, medial ½	
Med. brach cutaneous (C.8. T 1)	Skin, medial side of brachium	
Med. antibrach cutaneous (C 8, T 1)	Skin, antero-medial side of brachium and ulnar half of antibrachium	

PLATE IV

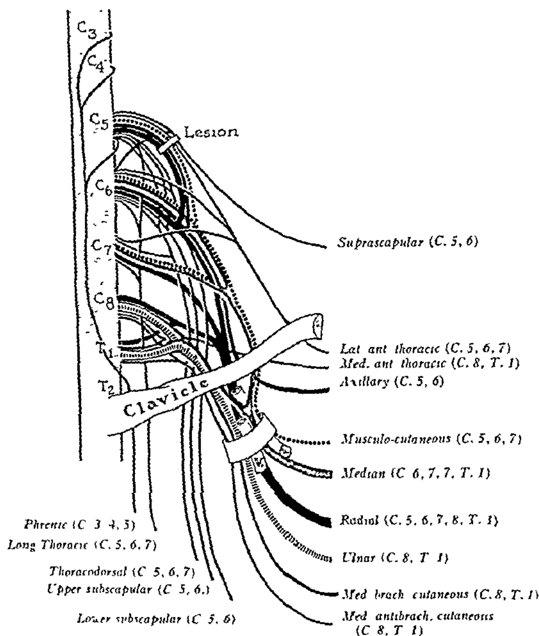


Diagram Which Shows a Lesion of the Brachial Plexus Which Produced a Weakness in the Supra- and Infraspinatus, Triceps, and Anconeus Muscles and a Paralysis of the Deltoid Muscle.

The chart on the opposite page shows the muscles which are weak or paralyzed and the nerves which innervate them. The combination of this chart and the diagram is useful in recording the diagnosis of a brachial plexus lesion.

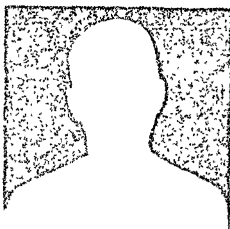
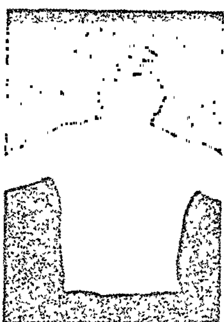
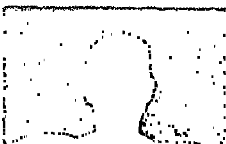
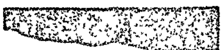
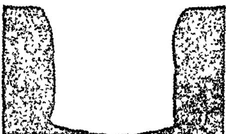
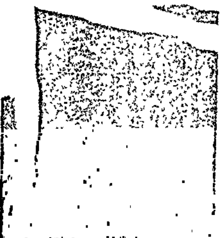


apart. The extent of the lesion depends upon the violence with which the nerve roots, cords, or trunks have been stretched and pulled, so that any degree of injury may be found in the same patient extending between the extremes of incomplete physiologic block and complete anatomic interruption of continuity.

Blunt injuries differ from stab or gunshot wounds in that the nerve fibers are not severed in a well-defined place, but are torn apart. The over-stretched funiculi may rupture at any point from the origin of the roots from the spinal cord to the most distal portion of the injury. Therefore, some fibers may be avulsed, others torn apart within the nerve trunks, and others over-stretched. The resulting clinical symptoms are those of complete and incomplete anatomic or physiologic lesions which cannot and should not be catalogued according to the early classification of brachial plexus injuries into the upper "Erb" or lower "Klumpke" types. We believe it is far more accurate and practical to determine the extent of the paralysis by careful, detailed examinations of the muscles and then place the level of the lesion by a knowledge of the innervation of those muscles. (Plate IV.) The progress of recovery can be followed more easily and a final evaluation of the residual lesion can be made accurately.

Rupture is most frequent in those portions of the plexus which are subjected to the greatest stretch, that is in the 5th and 6th cervical spinal nerves. Avulsion of the roots from the spinal cord is less frequent, and when it occurs, spinal cord symptoms are usually present. The lower roots or spinal nerves which contribute to the plexus; *i.e.*, cervical 7, 8 and thoracic 1, are less often injured and exhibit more evidence of spontaneous recovery. In stab or bullet wounds, the most exposed portions of the plexus are usually injured; *i.e.*, the 5th, 6th and 7th cervical nerves and the upper trunk of the plexus, in their course from the spinal column between the anterior and medial scalene muscles. In axillary and infraclavicular wounds, the individual nerves which arise from the cords of the plexus and the larger blood vessels are more likely to be injured. We have shown experimentally that bullet or other high velocity missiles have an additional concussive effect which may produce varying degrees of paralysis. Thus, immediately following injury, it is practically impossible to determine accurately the completeness of a brachial plexus injury until an interval has elapsed.

Injury to the contributing spinal nerves close to their exit from the intervertebral foramina or to the upper, middle and lower nerve trunks produces a segmental distribution of the motor and sensory disturbances. The paralyzed muscles fall into groups which correspond with the nerve roots from which they derive their innervation. Thus injury to 5th and 6th cervical spinal nerves and the

the distribution of the musculocutaneous and upper portions of the median nerves. Medial cord injuries affect the areas supplied by the ulnar, medial cutaneous of the arm and forearm and the lower portion of the median nerve. The radial, axillary, subscapular or thoracodorsal nerves are completely or partially involved depending on the location of the injury to the posterior cord. However, especially in blunt injuries, more than one component of the brachial plexus is involved; trunks or cords may be partly injured and compression and pull may be produced by hemorrhage, infection or an aneurysm. Subsequent extensive scar tissue formation tends to impair to various degrees many originally uninjured portions of the plexus and gives rise to disseminated and incomplete motor and sensory disturbances.

As a rule, immediately following the initial trauma to the brachial plexus the entire affected extremity is paralyzed and anesthetic. A physiologic lesion may recover spontaneously and fairly rapidly. In all types of brachial plexus injuries, physical therapy should be instituted at once and should be systematic and continuous over a long period of time, sometimes years. An attempt should be made to prevent contractures and deformities by proper splinting, massage and passive and active movements. Useful supplementary movements performed by unaffected muscles should be developed and when all evidence of regeneration and spontaneous recovery have ceased, or are unsatisfactory, surgical repair should be considered and carefully evaluated.

The indications for operation on the plexus vary greatly with each individual case and especially when the injury is due to blunt trauma. Signs of spontaneous recovery must be determined for the individual nerve roots and spinal nerves because the less damaged may regenerate and the more severely damaged ones may show no signs whatever of regeneration. Taylor,[1] who studied a large group of obstetrical brachial palsies, advised operation six to 12 months after the injury, depending upon the amount of spontaneous recovery that had taken place and the severity of the lesion. Finally, a large number of physicians consider brachial plexus lesions as irreparable and hopeless, while most neurological surgeons advise early surgical treatment. The outlook for recovery of function is much improved with prompt surgical repair. As time goes on, scar tissue becomes denser and constricts the plexus more and more, retarding and stopping all possiblities of nerve fiber regeneration. At the same time, muscle atrophy progresses, joint changes occur, and in old neglected cases the only method to obtain some function lies in plastic operations upon tendons or joints.

[1] Taylor, A. S.: The Brachial Birth Palsy. In Dean Lewis' Practice of Surgery Vol. III, 7, W. F. Prior and Co., Hagerstown, 1929.

Every attempt should be made to liberate the plexus from the the surrounding scar tissue by freeing and identifying the individual roots, trunks, cords and nerves, to resect the scar tissue and neuromas and to obtain end-to-end union of the ruptured nerves. This is often difficult, and at times impossible, because of the amount of scar tissue and contractions present, especially in old neglected lesions when the roots are avulsed from the spinal cord or torn within or near the intervertebral foramina. The experimental promise of properly used nerve grafts is particularly applicable to these injuries. The use of nerve grafts may be indicated where a loss of nerve substance is present and when end-to-end apposition is not possible. When autogenous grafts of the desired size are available, they provide the best type of graft which can be used. In some instances, resection of the middle third of the clavicle will make it possible to secure a better end-to-end apposition of the nerve of the severed nerve segment. At operation, the entire brachial plexus should be explored, the anterior scalene muscle and the clavicle divided if necessary, and proper electrical stimulation of the exposed nerves employed to detect the injured nerves. In old neglected lesions, the scar tissue may be so dense that even neurolysis becomes impossible and the segments of the severed nerves cannot be identified, or the plexus roots are avulsed from the spinal cord making suture impossible. In such cases, no improvement in function can be expected from the exploration of the brachial plexus. However, other surgical procedures should be used to obtain the optimal position of function for the extremity. The most gratifying result follows the shortening of tendons of paralyzed muscles or the implantation of tendons of normal muscles into the distal tendons of paralyzed muscles These plastic operations help to bring the hand, for example, into a position of optimal usefullness, or may bring about some dorsiflexion in an extremity in which all of the extensor muscles are completely atrophied and fibrosed.

The results of the surgical treatment of brachial plexus lesions are, on the whole, not as good as those of peripheral nerves. The distance between the site of the lesion and the muscles and skin involved makes new regeneration a slow and prolonged process. It is also obvious that neurolysis and nerve suture performed upon the brachial plexus cannot be done as extensively and accurately as upon a single peripheral nerve trunk Therefore, the end results should not be evaluated finally for several years after repair.

Cervical Rib.—Cervical ribs may produce symptoms from involvement of certain components of the brachial plexus. A cervical rib is a supernumerary rib arising from one of the cervical vertebræ, most commonly the seventh, but occasionally the sixth, and rarely the

fifth. Cervical ribs are present on both sides in about 80 per cent of all the cases, but only about 30 percent of these give bilateral symptoms. Though Galen and Vesalius described a cervical rib in detail, only 42 patients had been operated upon up until 1906.[1]

A cervical rib is a supernumerary rib arising from one of the cervical vertebræ, most commonly the seventh, but occasionally the sixth, and rarely the fifth. Cervical ribs are present on both sides in about 80 per cent of all of the cases, but only about 30 per cent of these give bilateral symptoms. Though Galen and Vesalius described a cervical rib in detail, only 42 cases had been operated upon up until 1906. We have operated upon 34 patients with cervical ribs, 19 of whom were females. With but 2 exceptions the ribs were present bilaterally, and in 5 instances the symptoms were bilateral.[1]

The characteristic syptoms are sensory disturbances, pain, muscular atrophy, and peripheral vascular changes. The pain is usually felt along the inner aspect of the upper extremity in an area which corresponds to the anatomical supply of the medial cutaneous, median and ulnar nerves. Rotation of the head, elevation of the chin and strong depression of the shoulder may produce a sudden sharp, lancinating pain but more often a dull, aching, heavy pain occurs after the day's work. This is particularly true after the usual routine of sweeping and dusting of a housewife's day or the overhead stretching movements performed by saleswomen. Hyperesthesias, anesthesias, but more often paresthesias which are described as tingling, burning and numbness, occur along the inner side of the forearm and in the hands and fingers.

Atrophy of the small hand muscles occurs slowly but progressively, and may produce an incomplete but still characteristic appearance and deformity of an ulnar nerve lesion. Muscular atrophy may occur in the small hand muscles supplied by the ulnar nerve or in more severe cases, a combined median-ulnar distribution may be present. (Fig. 163.)

Peripheral vascular lesions are rarely severe, and the most common symptoms are those of discoloration of the fingers and a marked sensitiveness of the fingers to changes in temperature. However, gangrene of one or more fingers has been reported and diminution in the volume of the radial pulse is quite common. Very often the pulse can be obliterated by elevating the chin and rotating the head so that the subclavian artery is compressed by the rib.

[1] By many, cervical ribs are regarded as a stigma of degeneration and features of an underlying neuropathic constitution. Many cases of syringomyelia have been associated with cervical ribs and in many instances ribs have been removed with the erroneous idea that they were the sole cause of the symptoms. Other congenital deformities, such as spina bifida and harelip, have also been reported as associated with cervical rib.

Wood-Jones[1] has attributed the formation of supernumerary ribs to the conflict in growth between the large plexus of cervical nerve roots and ribs Embryonic nerve trunks are much larger than the vertebræ and ribs and the oblique downward growth of these nerves normally inhibit the growth of cervical ribs so that only transverse processes are formed The extent of growth of a cervical rib is, therefore, dependent upon the resistance offered by a nerve trunk. To this conception, Todd has added that the blood-vessels also play an important rôle

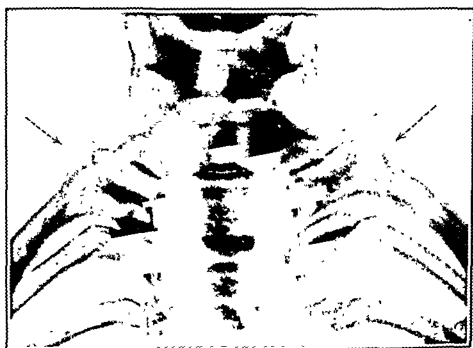


FIG 163 —Roentgen-ray film which shows bilateral cervical ribs, symptoms were present only upon the left side.

A complete cervical rib extends laterally from the seventh cervical vertebra, then turns forward and downward betwen the scalenus anticus and medius muscles to reach the costal cartilage of the first rib. The brachial plexus lies over it, and as it extends downward the subclavian artery arches backward and laterally over it The subclavian vein lies lower than the artery and lateral to the scalenus anticus which usually becomes attached to the rib as it pushes forward. Almost directly over the rib lie the suprascapular and transverse cervical arteries and above these vessels and running in

[1] Wood-Jones, F.: The Anatomy of Cervical Rib, Proc. Roy. Soc. Med, 6, 95, 1913.

the same general direction is the inferior belly of the omohyoid muscle. Over all, laterally and anteriorly, is the lower end of the sterno-cleido-mastoid muscle.

The surgical importance of the relationship of the nerve trunks and vessels, lying as they do between the scalenus anticus muscle and the cervical rib, has been emphasized by Adson and Coffey.[1] We have seen nerve trunks and the subclavian artery seriously compressed between an unyielding bony structure and a taut scalenus anticus.

The surgical removal of a cervical rib is indicated in the presence of the symptoms previously described, but occasionally a rib may be discovered quite accidentally by roentgen-ray examinations of the upper thorax without any accompanying symptoms. Not all ribs are calcified sufficiently to show definitely upon roentgen-ray films and in each of our patients the actual bony structure of the rib constituted only one-half of the total length. The remainder consisted of heavy, thick cartilage

For many years the removal of a cervical rib was a difficult, tedious surgical procedure attended only too often by a postoperative sensory loss and paralysis. The mid-cervical and post-brachial incisions involved considerable traction upon nerve trunks, and often it was difficult to disarticulate a rib because of the close proximity of the subclavian artery.

Adson's suggestion to employ an anterior transbrachial approach and his emphasis upon the important rôle of compression played by the scalenus anticus has simplified the surgical treatment of this condition tremendously. Very often, division of the tendinous attachment of the scalenus anticus and its immediate retraction, like the breaking of a taut violin string, makes actual removal of the rib seem unnecessary. As Adson says, "The ulnar nerve rides over a bony prominence where it is constantly subjected to motion without symptoms being provoked; there should be no more likelihood of symptoms arising in the brachial plexus when it merely lies on the cervical rib, with little or no motion and no traction on it."

We have, thus far, always removed the rib in addition to dividing the scalenus anticus. Relief from the pain and paresthesias has been immediate. Peripheral vascular changes improve slowly but progressively. When atrophy in the small hand muscles has been present for a year or longer, as it has in all of our patients, return to the normal volume cannot be expected with any degree of confidence in spite of physical therapy. There has been a slow but definite improvement in several of our patients, but this is something which cannot be more than a problematical result.

[1] Adson, A. W., and Coffey, J. R.: Cervical Rib, Ann. Surg., 85, 839, 1927.

Wood-Jones[1] has attributed the formation of supernumerary ribs to the conflict in growth between the large plexus of cervical nerve roots and ribs. Embryonic nerve trunks are much larger than the vertebræ and ribs and the oblique downward growth of these nerves normally inhibit the growth of cervical ribs so that only transverse processes are formed The extent of growth of a cervical rib is, therefore, dependent upon the resistance offered by a nerve trunk. To this conception, Todd has added that the blood-vessels also play an important rôle.

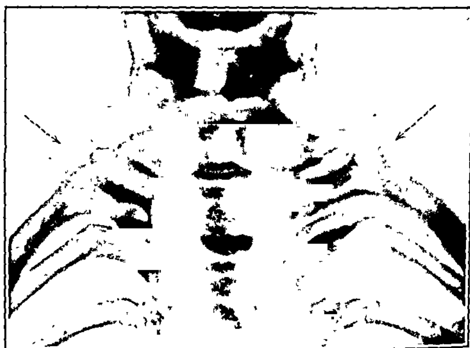


FIG. 163.—Roentgen-ray film which shows bilateral cervical ribs, symptoms were present only upon the left side.

A complete cervical rib extends laterally from the seventh cervical vertebra, then turns forward and downward betwen the scalenus anticus and medius muscles to reach the costal cartilage of the first rib. The brachial plexus lies over it, and as it extends downward the subclavian artery arches backward and laterally over it. The subclavian vein lies lower than the artery and lateral to the scalenus anticus which usually becomes attached to the rib as it pushes forward. Almost directly over the rib lie the suprascapular and transverse cervical arteries and above these vessels and running in

[1] Wood-Jones, F The Anatomy of Cervical Rib, Proc. Roy. Soc. Med, 6, 95, 1913.

the same general direction is the inferior belly of the omohyoid muscle. Over all, laterally and anteriorly, is the lower end of the sterno-cleido-mastoid muscle.

The surgical importance of the relationship of the nerve trunks and vessels, lying as they do between the scalenus anticus muscle and the cervical rib, has been emphasized by Adson and Coffey.[1] We have seen nerve trunks and the subclavian artery seriously compressed between an unyielding bony structure and a taut scalenus anticus.

The surgical removal of a cervical rib is indicated in the presence of the symptoms previously described, but occasionally a rib may be discovered quite accidentally by roentgen-ray examinations of the upper thorax without any accompanying symptoms. Not all ribs are calcified sufficiently to show definitely upon roentgen-ray films and in each of our patients the actual bony structure of the rib constituted only one-half of the total length. The remainder consisted of heavy, thick cartilage

For many years the removal of a cervical rib was a difficult, tedious surgical procedure attended only too often by a postoperative sensory loss and paralysis. The mid-cervical and post-brachial incisions involved considerable traction upon nerve trunks, and often it was difficult to disarticulate a rib because of the close proximity of the subclavian artery.

Adson's suggestion to employ an anterior transbrachial approach and his emphasis upon the important rôle of compression played by the scalenus anticus has simplified the surgical treatment of this condition tremendously. Very often, division of the tendinous attachment of the scalenus anticus and its immediate retraction, like the breaking of a taut violin string, makes actual removal of the rib seem unnecessary. As Adson says, "The ulnar nerve rides over a bony prominence where it is constantly subjected to motion without symptoms being provoked; there should be no more likelihood of symptoms arising in the brachial plexus when it merely lies on the cervical rib, with little or no motion and no traction on it."

We have, thus far, always removed the rib in addition to dividing the scalenus anticus Relief from the pain and paresthesias has been immediate. Peripheral vascular changes improve slowly but progressively. When atrophy in the small hand muscles has been present for a year or longer, as it has in all of our patients, return to the normal volume cannot be expected with any degree of confidence in spite of physical therapy. There has been a slow but definite improvement in several of our patients, but this is something which cannot be more than a problematical result.

[1] Adson, A. W., and Coffey, J. R.: Cervical Rib, Ann. Surg, 85, 839, 1927.

Anterior Scalene Syndrome.—As Naffziger and Grant[1] have said, whether or not patients have cervical ribs, similar mechanisms (embryological, anatomical, developmental, or occupational) may be present and produce symptoms.

Aching pain about the shoulder, stabbing or crushing pain in the fingers and hand, and numbness or tingling in the hand are the common symptoms of this syndrome. Pain may travel down the ulnar side of the arm or may spread over the shoulder, arm, neck, and even the face and scalp on the affected side. The pain is increased by any movement or position which stretches and tenses the anterior scalenus muscle and frequently to relieve their pain patients tilt their head toward the painful side. Flushing of the skin of the arm, trophic changes in the nails and skin, and a low blood-pressure in the arm on the affected side are common symptoms.

In all of our patients with this syndrome the anterior scalene muscle has been hard and tense and has seriously compressed the underlying subclavian artery and plexus. When its insertion is severed, the muscle retracts, and relief from the symptoms is immediate and lasting.

Sciatic Nerve.—Very often an injury of the sciatic nerve may not be classed as such if its peroneal or tibial division is injured alone in the thigh, as is not infrequently the case in a stab wound or bullet wound. We have recently seen such a case in which the scar tissue produced by a gunshot wound of the thigh involved only the peroneal division, which, as is often the case high in the thigh, was quite distinct and separated from the tibial division When the entire trunk is injured, the deformity is unmistakable. The foot dangles and drops as the patient walks, and it is brought down in a flail-like manner similar to the steppage gait of a peroneal nerve palsy. All of the muscles below the knee are paralyzed, and the patient is unable to stand on his heels or toes. The knee-jerk is always present and the Achilles' jerk is always absent, but muscle atrophy may be masked by the edema and infiltration of the tissues of the leg.

Sensation is diminished or lost over the entire foot with the exception of the inner border of the arch and the internal malleolus and the outer side of the leg to the knee. The overlap of adjacent uninjured nerves is considerable so that the upper level of the loss of pin prick may extend only to the middle third of the leg. As was noted in the median nerve, causalgia of the sciatic nerve occurs commonly The pain is referred to the sole and may be associated with a spasm of the muscles in the sole of the foot. Ulcerations of

[1] Naffziger, H., and Grant, W. T Neuritis of Brachial Plexus Mechanical in Origin; Scalenus Syndrome, Surg , Gynec. and Obst., 67, 722, 1938.



FIG. 164.—Note the atrophy of thigh and calf in patient with sciatic nerve lesion.



FIG. 165.—Peripheral nerve lesion. A sciatic nerve lesion illustrating the foot-drop and a hyperkeratosis which frequently appears in these lesions.

the plantar surface of the foot are common, and hyperkeratosis frequently occurs so that the skin over the dorsum of the foot may be covered by scales and crusts. (Figs 164 and 165.)

Peroneal Nerve.—The deformity produced by a peroneal nerve injury is analogous to that produced in a *radial nerve lesion*. The foot drop which occurs is complete and is often accompanied by a slight drop of the first phalanx of all of the toes. The steppage gait is characteristic, and the point of the foot is dropped and adducted. It is necessary to immobilize the knee of the patient when one examines for voluntary movements, but it is not difficult to observe



FIG 166 —Note the inability of patient to dorsi-flex the foot in a peroneal nerve lesion

that dorsiflexion of the foot and toes is impossible Adduction of the foot may be performed by the tibalis posticus muscle, and the distal phalanges of the toes may be extended by contraction of the interosseus tendon, but abduction of the foot is impossible It is important to remember that strong flexion of the toes may occasionally *result in inversion and slight dorsiflexion of the foot* by a mechanism similar to that which produces extension of the hand in radial nerve lesions when the flexors of the fingers are strongly contracted.

Sensation is lost over the dorsal surface of the foot and the anterior and lateral surfaces of the leg. In the foot sensation is lost to the base of the toes, extending *outward to a point between the fourth and* fifth toes and inward to the base of the first metatarsal bone Just as in radial nerve lesions, the isolated supply to pin prick sensibility may be extremely small and is usually represented by a narrow band

which extends from a point a little above the junction of the lower and middle thirds of the outer surface of the leg, diagonally across the dorsum of the foot to a point over the middle of the metatarsal bone of the great toes. (Figs. 166 and 167.)

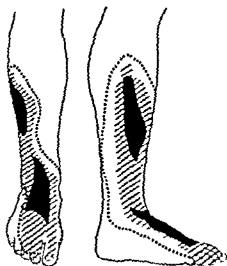


Fig. 167.—Sensory loss in a peroneal nerve lesion.

Tibial Nerve.—Lesions of the tibial nerve below the popliteal space are not common, and isolated lesions of the tibial division of the sciatic above that point are extremely rare. In one of our cases, the lesion was a severe laceration of the lower leg produced by a ploughshare. The most common motor disturbance is a paralysis of plantar flexion of the foot. True adduction of the foot is impossible; and if it can be performed, it is always accompanied by elevation of the foot and then is produced by contraction of the tibialis anticus. Flexion and separation of the toes are abolished, but the foot may be plantar flexed feebly by the peroneus longus. Walking is difficult, and the foot hangs down, is swollen, edematous, and discolored.

Sensation is lost over the sole of the foot except at the inner border, and frequently causalgia may occur, particularly in incomplete lesions of the tibial nerve. Trophic ulcers of the malleoli, heel, and toes, nail changes and edema are common; and in our own case an indolent ulcer of the heel was the most disturbing symptom which kept the patient from working on his farm. (Figs. 168 and 169.)

Of the remaining peripheral nerves, the *axillary*, *musculocutaneous*, and *femoral* nerves are occasionally injured. We have had occasion to see several patients with an axillary nerve lesion in consultation but have had none in our own series. Commonly observed in brachial plexus injuries, where it may exist as a residual lesion, an isolated axillary nerve paralysis is rare but may

result from fracture of the head of the humerus and subglenoid dislocations. Paralysis of the deltoid muscle with loss of abduction of the *arm from the* side is the most characteristic symptom. Because the physiology of the movements of muscles about the shoulder is very complex, supplementary movements which produce abduction of the arm may be developed to the point where it is extremely difficult to detect a loss of that movement. However, the pronounced atrophy of the deltoid muscle, which causes the acromial end of the clavicle to jut out prominently, is unmistakable. The sensory loss in axillary nerve paralysis occupies an area over the deltoid prominence.



FIG 168 —Note inability of patient to flex foot and toes in a tibial nerve lesion

The *musculocutaneous* nerve, which innervates the biceps muscle, is rarely injured alone but is *commonly involved in* brachial plexus injuries Flexion at the elbow joint with the forearm in supination is impossible, but the patient learns quickly to flex his forearm by use of the brachioradialis muscle so that flexion occurs with the forearm midway between supination and pronation The sensory loss is to be found over the radial side of the volar surface of the forearm. We examined three paratroopers in the European Theatre of Operations who had received a direct injury to the musculocutaneous nerve when their parachute harness slipped during their descent and produced severe

traction acrosss their upper arm. All recovered spontaneously without operation.

The *femoral* nerve may become involved by tumors, operative wounds, psoas abscess, or in prolonged gynecological operations in which the thighs are strongly abducted. If all of the branches of the nerve trunk are involved, the hip joint cannot be flexed, and the leg cannot be extended. Walking becomes difficult, and frequently the leg gives way at the knee if only the terminal branches to the extensor muscles of the leg are paralyzed. Atrophy is present in the thigh, and there may be a loss of sensation over the outer surface of the thigh and the inner surface of the leg



Fig. 169 —Note ulcer on sole of foot in a tibial nerve lesion.

Electrical Reactions.—Though the particular muscles involved and the extent of the sensory loss vary with the particular nerve injured, in every case the muscles so paralyzed react abnormally to electrical stimulation. The muscles will not react to the faradic current; and although early after an injury they are hyperexcitable to the galvanic current, this reaction also disappears after about two weeks. Then the quick twitch of the normal muscle is replaced by a slow contraction of the muscle sarcoplasm which is the only contractile tissue remaining in the muscle. While this is a very simple statement of the "reaction of degeneration," it is a practical means of using a rather confusing law which states that in the normal muscle the cathodal closing contraction is greater than the anodal

closing contraction; but in the muscle whose nerve supply has been severed, *this condition is reversed.*

Due to the impetus provided by the large number of peripheral nerve injuries which have occurred during World War II, Pollock and his *associates*[1] *have contributed important data to the question* of electrodiagnosis. In the main, Erb's description of the characteristics of complete reaction of degeneration have been confirmed *They emphasize that faradic stimulation* is effective throughout degeneration, denervation and regeneration, providing sufficient amperage is employed. The changes of response to galvanic stimulation of "infinite time" which characterize complete reaction of degeneration are (1) hyperirritability of muscle to galvanic stimuli, (2) sluggishness of relaxation of the contraction wave, (3) lessening of the ratio between amperage necessary to produce tetanus and rheobase to almost unity and (4) increase of efficacy of the anodal closing stimulus to equality with the cathodal closing stimulus They state that the unequivocal signs of recovery are (1) a sudden increase in rheobase, (2) an increase of ratio between the amperage necessary to produce tetanus and rheobase and (3) an increase in efficacy of the cathodal closing stimulus as compared with the anodal closing stimulus. A very high ratio or a very high threshold amperage are indicative of regeneration and these electrical signs of recovery antedate the recovery of motion and sensation by many weeks.

The characteristics of response to electrical stimuli for various states of muscle are summarized by Pollock as follows:

Normal.—A chronaxia equal to a small fraction of a millisecond, a rheobase ratio greater than 1.0 in the majority of cases, a tetanus ratio of from 3 2 to 9.5, a continuous strength-duration curve, *reaction to faradic stimuli and slight facilitation with an optimum* interval as determined by repetitive stimuli.

Denervation – A chronaxia greater than 15 milliseconds, a rheobase ratio of 1.0 or less in a considerable number of cases, a tetanus ratio of 1.0, a continuous strength-duration curve, no reaction to faradic stimuli, and pronounced facilitation with a high interval ratio, as determined by repetitive stimuli.

Early Degeneration.—A high tetanus ratio at a time before regeneration could have occurred, followed later by a tetanus ratio of 1.0

[1] Pollock, L. J, Golseth, J G, Arieff, A. J., Sherman, I C, Schiller, M A., and Tigay, E L.: "Reaction of Degeneration in Electrodiagnosis of Experimental Peripheral Nerve Lesions," War Medicine, 7, 275. 1945. Pollock, L. J, Golseth, J. G, Arieff, A J, and Mayfield, Frank J.: "Electrodiagnosis by Means of Progressive Currents of Long Duration," Surg, Gynec. and Obst., 81, 192, 1945.

Pollock, L J., *et al* "Electro-diagnosis of Lesions of Peripheral Nerves in Man," Arch Neur and Psych, 60, 1, 1948.

PLATE V

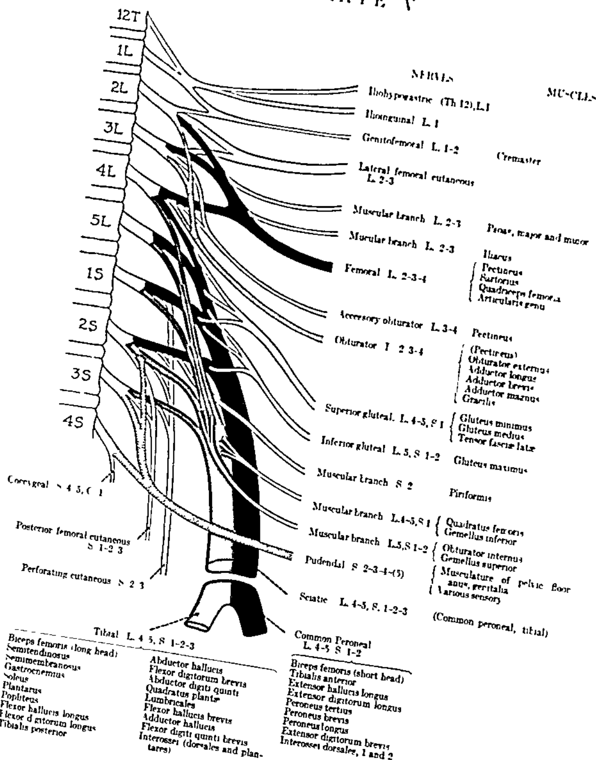


DIAGRAM OF LUMBO-SACRAL PLEXUS

Early Regeneration.—A high tetanus ratio, increase of rheobase, and at times, a shortening of chronaxia.

Maturation.—A chronaxia of 15 milliseconds or less, a high tetanus ratio; response to faradism and a flat curve, as determined by repetitive stimuli.

Partial Lesion.—An interval since injury of less than 100 per cent neurotization time, but one within which denervation should have occurred, a tetanus ratio of about 2.0, a chronaxia of less than 15 milliseconds, reaction to faradic stimulation, and a flat strength-interval curve, these characteristics being found together or alone.

The electromyograph reaches a high degree of accuracy in the diagnosis and prognosis of peripheral nerve injuries. A completely severed peripheral nerve will show, approximately eighteen days after injury denervation fibrillation voltages from all areas of the muscle tested. No discrete motor unit voltages are produced from any portions of the affected muscles by voluntary contractions. As nerve regeneration occurs, beginning motor unit contractions may be seen in the paralyzed muscles. Often these electromyographic phenomena can be seen long before clinical evidence of motion occurs and gives assurance that the nerve suture is intact and reinnervation is occurring.[1] In spite of the evidence of complete muscle paralysis, the electromyograph may be able, on finding motor unit contractions, to show that intact nerve fibers exist and, therefore, that the chances of spontaneous regeneration are good.

Differential Diagnosis.—This combination of motor and sensory loss and change in the reactions to electrical stimulation readily differentiate a nerve lesion from severed tendons, ischemic paralysis, or fibrotic and inflammatory reactions. The segmental distribution of the paralysis and dissociation of sensory loss aid in differentiating intramedullary spinal cord lesions, such as syringomyelia and tumors. The disturbances of motion and sensation which are found in hysteria never conform to the known organic symptoms which accompany specific peripheral nerve lesions. For example, one of our patients had received a trifling injury to the arm while at work and maintained that he was unable to flex the wrist or any of his fingers. Yet when asked to do so, he could appose his thumb and little finger and could grasp a piece of paper tightly between the thumb and index finger. The first movement was a test for median nerve function and the latter for the ulnar nerve. Consequently, it could be concluded that his combination of symptoms could not be the result of a combined median and ulnar nerve paralysis, which was suspected because of the loss of motion in his wrist and fingers. Moreover,

[1] Woods, W. W., and Shea, P. A.: "The Value of Electromyography in Neurology and Neurosurgery," Jour. Neurosurg., 8, 595, 1951.

the loss of sensation in a functional lesion is likely to be glove type or may resemble some geometric figure. In addition, the purely objective reactions to the galvanic current quickly rule out an organic lesion.

We have observed 2 patients in whom contractures and loss of function in the hand occurred, and the site of the wound was at a point on the body far removed. In both instances the hands were cold and cyanotic; the amplitude of the pulse was diminished; perspiration was excessive, and the skin was macerated. Babinski and Froment[1] designated this clinical entity as a *reflex nervous* disturbance and later as a physiopathic affection, while Meige and Benisty[2] named the condition "congealed hand." Carefully directed and persistent physical and psychotherapeutic treatments resulted *in the cure of both of these patients.*

SURGICAL INDICATIONS

Many attempts have been made to describe the syndromes produced by physiological and anatomical interruption of the nerve fibers, but *there is no way by which complete anatomical and physiological interruption can be differentiated.* We can determine whether *or not the loss of function is complete or incomplete. If it is incomplete*, anatomical division cannot be present except as a lateral notch. In the presence of complete physiological interruption, one may say that an anatomical lesion does not exist only when subsequent examinations show some evidence of return of function. Complete paralysis of all of the muscles supplied by the nerve below the level of the lesion, loss of normal electrical reactions in the muscles, extensive and rapid muscle atrophy, and loss of sensation in the area of isolated supply of the nerve may be accepted as evidence of complete interruption of nerve function. Any one of these signs alone may lead to rather wide errors in diagnosis.

Several factors have been of distinct help to us in deciding upon a course of action when it was obvious that complete interruption of nerve function was present. For example, stab or lacerated wounds are more likely to produce an anatomical division, but a blunt force applied over the course of a nerve may produce only physiological interruption without severance of continuity. If this force be applied to the nerve in a part of its course where it is well protected by muscles, the injury may be less serious than if it is over a more superficial portion of the nerve. Fractures of bone may produce

[illegible] et troubles nerveux [illegible] 7 pp., 1917.

[illegible] s aux blessures de Nerfs periphenques, Rev neurol., Paris, 33, 631, 1916

an anatomical section, or the nerve may be involved in bony callus, and the symptoms may first appear when the cast is removed. Of course, in many instances of anatomical section we have been able to feel the distinct neuromas upon the divided nerve ends.

We have had the opportunity of observing several cases in which the clinical signs of complete nerve interruption were present without anatomical severance of the nerve and without compression of the nerve. These occurred as the result of injuries by small fragments of the 20 mm. Oerlikon shell which have a high velocity and which passed through the tissues in close proximity to the nerve. The symptoms resulted from the concussive effect of the high velocity fragment. It has been shown experimentally that the microscopic appearance of degeneration from concussion thus produced is severe and differs considerably from that of the common Wallerian degeneration. It extends a considerable distance centralward from the site of injury. Regeneration is slow and imperfect and particularly in war wounds this factor must always be kept in mind as an explanation for poor functional recovery.

It is just this type of case, however, which calls for good surgical judgment as to whether the nerve injury should be operated upon or treated conservatively until it is certain that no sign of recovery is occurring. Practically, in the past and in a general way, if there was no evidence of regeneration from four to five months after complete paralysis of the radial nerve, surgical exploration was advised. Similarly, the fifth month was considered as the maximum time to wait for spontaneous recovery in complete paralysis of the ulnar nerve and the sixth month for median nerve lesions. With the recent developments in electrodiagnosis described just previously, a more accurate method for making a decision for or against operation has been provided and valuable time will not be wasted.

It cannot be insisted upon too vigorously that the ideal time for the repair of a peripheral nerve injury is immediately after it has been received, granting that all conditions are favorable. For example, a lacerated wound at the wrist produced by a knife or piece of glass should be cleansed surgically and thoroughly with soap and water; the tendons repaired and the nerve ends apposed accurately with the finest silk sutures available and the wound closed. The longer the interval between the receipt of the wound and its surgical treatment, the more likely is infection to occur, but it has been shown that the meticulous surgical cleansing of a potentially or grossly infected wound plus the judicious use of the sulfonamides and antibiotics, within thirty-six to forty-eight hours, will permit of end-to-end apposition of divided nerve ends and, in the majority of instances, healing of the wound in the soft tissues by primary

intention. Granted, that the wound may suppurate in spite of these carefully carried out surgical steps, the nerve ends are in apposition and infection does not interfere with the regeneration of the neuraxons unless it gains access to the nerve fascicles. That is to say, infection in the surrounding soft tissues does not interfere with regeneration.

In the past, it was thought that an infected wound should not be reopened after it had healed for at least four to six months because of the possibility of relighting the infection. Thus, under such conditions divided peripheral nerves could not be repaired as quickly as they should. With the present day use of the sulfonamides and penicillin, this type of wound which had healed following infection has been opened, the nerve sutured, the wound closed, and healing has occurred. Experiences with war wounds of extremities received during the North African campaign showed that this procedure was feasible and successful.

The important thing to remember is that the effector mechanisms, that is, the muscles, the tendons and the joints become atrophied, fibrotic and shortened, or lengthened, as the result of denervation and disuse. It is these organs which produce the functional result because regeneration of neuraxons will occur whenever the nerve ends are united. Therefore, there can be no sound surgical reason for the deliberate policy of delaying a nerve suture. Repair must be done as soon after division of the nerve as is surgically possible.

It has also been proven experimentally that the sulfonamides introduced into a wound locally or administered systemically in proper dosage will not interfere with the regeneration of nerve fibers. The advantage of obtaining apposition of the nerve ends far exceeds the small amount of adhesions which accompany their use in the surrounding soft tissues Therefore, if an extensive soft tissue injury has occurred and it becomes necessary to transport the patient a considerable distance with the elapse of a number of hours before proper definitive surgical treatment can be effected, immediate mechanical cleansing of the wound with soap and water, the introduction of sulfonamides locally into the wound and their administration systemically will not interfere with nerve regeneration but, on the contrary, will make it possible to obtain nerve apposition earlier.

In war wounds, particularly, there are often concomitant injuries of the vessels of an extremity. The resulting anemia of the tissues results naturally in the symptoms of a peripheral nerve injury even though the nerve trunk has suffered no direct interruption of its anatomical or physiological continuity. The total character of the sensory loss and the absence of vessel pulsations establish the nature of the lesion, for which an immediate paravertebral sympathectomy

should be performed. However, it is impossible to determine immediately whether or not any one nerve trunk has been directly injured.

Often it requires good surgical judgment to decide whether or not a fracture should be treated without regard to the nerve lesion, whether they should be treated simultaneously or whether the nerve ends should first be apposed. Each case is an individual problem but in general the bone fracture should receive primary consideration.

Likewise, there is often a large loss of soft tissue in an extremity which requires repair by skin grafting. If the nerve ends can be apposed without the necessity of placing the extremity in such a posture that the skin graft cannot be performed, then the area should be covered with skin and later the nerve ends should be apposed. Often, however, both procedures can be performed simultaneously.

SURGICAL TECHNIQUE

Contrary to the general scope of this book, more detail concerning the technique of repairing peripheral nerve injuries is given in the hope that it may be of aid to the general surgeon who may be called upon infrequently to operate upon severed tendons and nerves.

In speaking of nerve suture we mean, of course, the direct end-to-end apposition of the divided nerve ends. It has been proven both experimentally and clinically that this method of repair, properly performed, may be depended upon to obtain successful results. Other methods of nerve repair[1] have been described mainly to overcome large defects in the substance of the nerve trunk. They

[1] These include nerve implants, nerve crossing, nerve flaps, suture ádistance, tubular suture, cable grafts, and nerve transplants. Of these methods, autogenous nerve transplants, or grafts, offer the most satisfactory method to obtain a successful result However, homogenous nerve grafts which have been freshly removed from the donor have not been used sufficiently in the human to state definitely that they are failures

To obtain in man the good results which are seen in nerve grafting in animals, the graft must be implanted only between fully viable, not severely traumatized, nerve ends. Grafting should not be accompanied by excessive stretching of the nerve and our animal experiences teach us that autogenous grafts are preferable to homogenous grafts

Grafts should have an optimum of blood supply in order to survive or become revitalized The survival and good revitalization of a graft depends upon its length We have always found experimentally that necrosis occurred more commonly in the central area of the graft and that shorter grafts have a better chance to become revascularized from their ends than do longer grafts. In view of our animal experience, it is hard to believe that dead endoneurial tubes should offer [illegible]
chances for [illegible]
have at leas [illegible]
embryos or [illegible]
immature or [illegible] may show much less tendency to dense fibrous reaction than that of adult tissue.

are obviously operations of second choice. For this reason, we have always made the greatest effort to overcome defects in substance by transposition of the nerve from its normal anatomical course to a shorter one, and flexing or extending joints adjacent to the defect in order to obtain direct end-to-end union. It is surprising to note that defects of 3 to 5 inches can be overcome in these ways. Two or three weeks after the suture has been performed, the extremity can be gradually returned to a normal position. This gradual stretching of the nerve trunk may retard signs of recovery of function. Experimental work has been done[1] to show how much elongation of nerves under degrees of tension may produce histological changes in the nerves. Under a strain of 22 per cent of the length of the nerve, the perineurium may split longitudinally with herniation of nerve fibers in the formation of pseudoneuromas. Transverse tears of nerve fibers and endoneurium may occur with an elongation of 20 per cent of the length of the nerve. Tears of the myelinated fibers at any one given point occur with a 10 per cent stretch and individual fasiculi may be ruptured at different points with a strain of 6 per cent whereas minimal single fiber tears occur with a strain of 4.7 per cent. It may be said that a nerve trunk must not be "stretched" more than 6 per cent of its mobilized length. Even though the wound does not produce a loss of nerve substance, in practically every case, a gap of several inches must be bridged after the neuromas have been resected from the nerve ends. This is only another argument for the early suture of a divided nerve.

The question of how nerve fibers regenerate after division has been the subject of innumerable contributions, among the most important of which have been by Howell and Huber[2] and Ranson.[3] The basic fact which underlies the entire problem is that the portion of a nerve fiber separated from its cell of origin degenerates and that portion which remains attached will show attempts to regenerate. This accounts for the characteristic histological appearance which has been described in the proximal and distal segments at varying periods during degeneration and regeneration. Evidence of degeneration in the motor and sensory nerve endings, as well as the neuraxones, is present *immediately after nerve section and proceeds* until the axons of sensory end organs disappear completely.

The importance of the mesodermal tissue in nerve regeneration has clearly been demonstrated by the microscopic study of nerve sections stained with

[1] Lin, C. T., Benda, C. E., and Lewey, F. H.: "Tensile Strength of Human Nerves: An Experimental, Physical and Histologic Study." Arch. Neur. and Psychiat., 59, 322, 1948.

[2] Howell, W. H., and Huber, G. C.: A Physical, Histological and Clinical Study of the Degeneration and Regeneration in Peripheral Nerve Fibers After Severance of Their Connections With the Nerve Centers. Jour. Physiol., 13, 335, 1892.

[3] Ranson, S. W.: Degeneration and Regeneration of Nerve Fibers. Jour. Comp Neurol., 22, 487, 1912.

silver and counterstained with fuchsin.[1] This stain makes it possible to study simultaneously the reactions which take place in the nerve fibers, the endoneurium, the Schwann cells, the perineurium, the epineurium and the capillaries following section and repair of the nerve by end-to-end suture or the transplantation of an autogenous or homogenous graft.

The mesodermal tissue is the first to react following any nerve injury by proliferating. This proliferation takes place at the site of the lesion and from there on into the gap between the severed nerve segments, in the degenerated distal nerve segment, in the perineurium and the epineurium of the central as well as the distal nerve segment. Regenerating nerve fibers follow the path of proliferating histiocytes and collagenous fibers. Wherever this proliferative [illegible] segment of a cut nerve exerts a chemotropic influence on regenerating nerve fibers growing out of the end of the central segment.

The mesodermal tissue plays a primary role in the organization of autogenous and homogenous grafts in laying down a scaffolding which is followed by the regenerating nerve fibers. Depending upon the degree of survival of the mesodermal elements in the graft and the degree of necrosis which took place, this scaffolding follows the original nerve structure or becomes irregular, deviating, and confused in its course and thus influences the course and the efficacy of the regenerating nerve fibers.

A very definite contusion effect within the nerve is found following gunshot injuries which do not interrupt the continuity of the nerve. Extensive traumatic damage is present for a long distance centrally and peripherally from the site of trauma. It leads to a process of necrobiosis and dissolution of the ectodermal elements with survival of the mesodermal elements, and results in an obliteration of the endoneural structure and a marked confusion in the course of the regenerating mesodermal fibers and nerve fibers.

The downgrowing axons do not exhibit any special selectivity as they reach the distal segment. Motor fibers may enter the neurilemma sheath formerly occupied by sensory fibers and *vice versa*. Under such conditions functional regeneration is probably impossible although there are some observations which tend to show that the nerve endings attempt to change their structural character. This fact emphasizes the importance we have always attached to the care which must be taken to obtain an accurate end-to-end apposition *without rotation* of either end of the nerve trunk. No doubt it is also responsible for many poor results which have followed nerve suture.

Peripheral nerve operations require patience and meticulous attention to detail so that no mechanical obstacle under the control of the surgeon may hinder regeneration. It is our practice to sterilize the skin of the extremity well above and below the proposed limits of the incision. The operative sheeting should be arranged so that the extremity may be observed and manipulated freely without contamination of the surgical field. It is essential to be able to observe

[1] Davis, L., Perret, G., Hiller, F., and Carroll, W.: Experimental Studies in Peripheral Nerve Surgery, Surg., Gynec. and Obst., 79, 245, 1944; 80, 35, and 81, 302, 1945.

all muscle movements which may be produced by stimulation of the nerve trunk and to be able to move the extremity into any desired position.

The incision should extend well above and below the probable site of injury. The normal trunk should be exposed and the dissection carried toward the lesion. The contraction of scar tissue, the bulbous neuroma on the central end of the nerve, the distorted appearance of the peripheral end, and the change of the normal anatomical relations all serve to make identification difficult. A careful, clean, sharp dissection should be confined as nearly as possible to normal lines of cleavage. Bleeding vessels should be grasped individually and not in the center of a mass of tissue which surrounds them. *Sponging should be done with gauze or cotton pledgets* moistened in normal saline solution. The nerve should be protected at all times by wet cotton sponges If it is necessary to handle the nerve during its exposure, a narrow moistened cotton tape beneath it will prove advantageous.

After the nerve and its neuromatous ends have been isolated and freed and before an attempt is made to approximate the ends, the field of operation should be made as bloodless as is possible In our hands the most practical method of constriction has been to use a blood-pressure cuff. This is unnecessary, however, if careful attention is paid to bleeding vessels when the incision is made and the nerve is exposed Many times, we have felt somewhat uneasy at the task of securing hemostasis after the suture line in the nerve has been *completed and this is what is necessary if a tourniquet* is used to control bleeding during operation.

When the normal nerve trunk has been identified above and below the lesion and before it has been handled, we place a single silk suture through the epineurium exactly in the midline. This prevents accidental torsion of the nerve from its normal axis and, as has been stated, it is important to obtain an approximation of the nerve ends in as nearly the normal funicular apposition as is possible. With a sharp, safety razor blade the neuroma on the central end should be sectioned successively at intervals of a millimeter until *the dense scar tissue has been passed and normal* nerve structure is identified. The hard, gritty sensation obtained in cutting through the neuroma is in contrast to that experienced when normal nerve tissue is encountered The fresh normal nerve end is soft and resembles the end of a cut telephone wire cable with bundles of nerve fibers bulging out beyond the end of the nerve sheath In a similar manner scar tissue should be removed from the distal end The resection of the neuromas must be carried out *thoroughly* because the presence of scar tissue in the nerve ends will interfere seriously

with the chances of obtaining a successful result. Often after the neuroma has been sectioned from the central end, bleeding occurs from a small vessel in the epineurium. Usually this may be controlled by a warm cotton pledget, but occasionally it is necessary to crush the vessel with a mosquito hemostat.

The sutures placed through the nerve sheath to prevent torsion should be held in the position in which they were placed. The first suture of fine silk is placed through the epineurium only of the central and distal segments. It should not pass between or in any way constrict the nerve funiculi. Similar sutures should be passed in the medial, lateral, and posterior quadrants of the nerve. The latter may be exposed by pulling the lateral quadrant suture to the medial side. By exerting simultaneous traction upon all four of these sutures, the nerve ends are brought into approximation, and each suture is tied firmly but not so tightly that the ends of the nerve fibers are overlapped. Intermediate sutures should then be placed in the nerve sheath around the circumference of the nerve trunk so that none of the nerve fibers gapes through the line of suture. The finest corticelli black silk[1] is separated into its component strands, and each of these serves as a suture after it has been passed through vaseline gauze.

The united nerve should then be allowed to drop into its bed. Normal untraumatized muscle bellies are preferable as a protection for the sutured nerve. If a large amount of scar tissue is present in the original bed, adjacent normal muscle may be drawn together under the nerve. If this is impossible, the nerve may be placed in a slightly different position without interfering with its function, or a flap of superficial fat may be swung beneath the suture line. Tantalum foil has been used by some surgeons to wrap around the site of the suture line, particularly in wounds in which normal muscle tissue is difficult to find. It has been shown experimentally that vascularization of the suture line and particularly of nerve transplants comes from the surrounding tissue. Any material wrapped about a nerve trunk prevents the ingrowth of vessels and may constrict the suture line seriously.

The majority of the smaller defects may be overcome by careful mobilization of the nerve trunk below and above the lesion. This may mean only freeing the nerve from its normal bed between muscle bellies. It is important to avoid destruction of even the smallest

[1] Sargent and Greenfield have shown that although silk gives rise to a slight tissue reaction, chromic or other [illegible]
reaction. The tensile stren [illegible]
and yet the reaction of the [illegible]
Experimental Investigation [illegible] Materials Used for Nerve Suture, Brit. Med. Jour., ii, 407, 1919.

muscular branch. Often these branches may be dissected free so that their mobilization will add to that obtained in the nerve trunk. As we have said, in a large number of cases nerve ends may be *approximated by utilizing the relaxation of the nerve trunk* which may be obtained by changing the position of the governing joints. For example, flexion of the wrist will make it possible to approximate the ends of the median and ulnar nerves in the forearm though a large defect be present. After the suture has been completed, the joint must be kept in the position of relaxation by a light restraining splint for at least two weeks.

In the arm particularly, *the anatomical course of the nerves may* be shortened considerably by transposition from the dorsal to the ventral surface. It is possible to gain several inches by transposition of the ulnar nerve from behind the inner condyle of the humerus to the flexor surface of the elbow. The radial nerve may be transposed to the anterior surface of the humerus with a gain of almost 3 inches. The median nerve has a more direct course, but some lengthening may be obtained by freeing its muscular branches in the forearm and transposing the nerve to a more superficial position. In some instances the defect to be bridged before end-to-end suture is possible may be so great that none of these methods will suffice. Under *such conditions the central and distal ends should be mobilized* as much as possible; the neighboring joints should be placed in position to afford the greatest relaxation possible, and if necessary, transposition should be done. Then the neuromas should be sutured together. After a week's interval the position of the extremity is gradually changed so that the nerve trunk is gradually lengthened by stretching. When the extremity has been fully extended, the second operation is performed. The neuromas are resected and approximation is obtained.

In one of our patients the radial nerve was only partially injured by a stab wound in the middle of the arm. The point of the blade had injured the nerve sufficiently to produce a lateral neuroma, and the infection which had followed caused scar tissue sufficient to compress the remaining normal fibers. The lateral neuroma was resected, without dividing the trunk entirely, after a careful external neurolysis had been done.

Injuries of the palm of the hand very frequently injure the digital branches of the median and ulnar nerves. These injuries are quite commonly produced by broken porcelain *water faucet handles*. These nerves are important because they innervate the lumbrical, interossei and thenar and hypothenar muscles as well as supplying sensation to the palm and fingers. They lie just beneath the palmar

aponeurosis and superficial to the tendons. Unless they are identified early in the operation, they may become damaged irreparably.

One should acquaint himself thoroughly with the anatomical relations of the involved nerve. One or two points have impressed us as having unusual surgical importance. Except for the tendon of the palmaris longus, the median nerve is the most superficial structure beneath the deep fascia of the wrist. In the forearm it lies between the superficial and deep flexor tendons. One may be confused in attempting to find the distal end of the median nerve at the wrist if it is not remembered that it lies directly beneath the transverse carpal ligament. On the contrary, the ulnar nerve at that level lies under the edge of the flexor carpi ulnaris tendon and passes into the palm of the hand superficial to the transverse carpal ligament. When the lesion lies at this low level, it must be remembered that the ulnar divides into a deep and superficial branch. Care must be exercised to make sure that both branches are sutured.

From our experimental and clinical investigations, fresh autogenous grafts which approximate the diameter of the injured nerve are practical and the functional end results are good. Fresh homogenous adult or young nerve grafts have not been used and studied sufficiently in patients to nullify the encouragement which animal experiments provide. The overgrowth of mesodermal tissue is more marked in homogenous than in autogenous grafts. However, when vascularization is possible from the surrounding tissues, fresh homogenous grafts remain viable in humans.

REHABILITATION

It is unfortunate that many times nerve ends are sutured and no thought is given to what we consider to be at least of as great importance: *carefully supervised and persistent physical therapeutic aftercare.* If the paralyzed muscles are allowed to shorten and contract or the joints become ankylosed or fibrosed, it is of little value to have sutured a nerve and have its fibers regenerate. Every effort must be directed toward restoration of the normal physiological function of the paralyzed muscles. Although it is somewhat difficult to measure accurately, we are firmly convinced that those patients who receive physical therapy show evidences of recovery of function much earlier, and the degree of recovery is much greater.

There is a definite increase of connective tissue in a muscle as early as three weeks after section of its nerve. Part of the late contractures are due to shrinkage of this newly formed, soft and extensible tissue. Active or passive movements of the muscle will stretch the developing connective tissue fibers so that when they

shrink there may be less tendency to a contracture. Passive exercises may be carried out very well in conjunction with massage.

Physical therapy should be carried out by individuals who fully realize the ease with which atrophied, denervated muscles may be injured by rough methods and fatigue. In a great many cases better results are obtained by simple devices and games which effect an unconscious and effortless exercise of the contracted muscles or ankylosed joints. We have found that most individuals require a definite goal to accomplish in each type of active exercise instituted to maintain their enthusiasm during the long period of physical treatment which is necessary.

Likewise, it must be emphasized that only those methods of electrotherapy are useful in the postoperative treatment of these patients which produce contractions of the denervated muscle. Judiciously used, electrotherapy may conserve the volume and nutrition of the muscle and keeps the fibers functionally adequate for voluntary movement when regeneration has progressed sufficiently.[1]

Just as the purpose of massage, exercises, and electrotherapy is to maintain the nutrition of the denervated muscles, the first aim of *mechanical splints* should be to prevent overstretching of the paralyzed muscles, to restrict shortening of the normal antagonistic muscles, and to maintain as nearly as possible the normal range of the joints and tendons of the affected extremity. A single splint for each type of peripheral nerve injury cannot be recommended dogmatically. Each case is an individual problem, and the splint should be devised to fit the patient and his injury. The ideal type of splint should be light, simple, easily applied and removed, inexpensive,

[1] Obviously, the faradic current cannot be used because the duration of each shock is too short in relation to the changed chronaxie of the nerve and muscle. Therefore, galvanic current must be used This should be employed in its simple form of a continuous current or in the form of a sinusoidal current of various types [illegible] n be used to be obtained [illegible]

[illegible] s which indicates that electrical stimulation does nothing to aid in recovery of function but that massage and motion are a distinct help. Chor, H. *et al.* Atrophy and Regeneration of the Gastrocnemius-Soleus Muscles, Effects of Physical Therapy in the Monkey Following Section and Suture of the Sciatic Nerve, Jour. Am. Med Assn, **113**, 1029, 1939

Daily application of galvanic exercise with a current strength sufficient to elicit vigorous contractions of the muscles was found to delay and diminish atrophy in denervated and re-innervated rabbit muscle. The most satisfactory results were obtained with a daily exercise of twenty to thirty minutes duration The effect was greater the earlier the treatment was started following denervation

Gutmann, E, and Guttmann, L. "*The Effect of Galvanic Exercise on Denervated and Re-innervated Muscles in the Rabbit,*" Jour Neur., Neurosurgery and Psych., 7, 7, 1944

and as inconspicuous as possible. The character of the splint required *depends* also upon the time which has elapsed between the original injury and the institution of treatment. In our experience, the most difficult and complicated problem is offered by the patient who comes for treatment long after the original injury with fibrosed joints and stretched, atrophied muscles. A splint should be so devised as to maintain the part in as nearly as possible its normal position of function, and traction upon ankylosed extended phalangeal joints should be elastic and easily varied as conditions change.

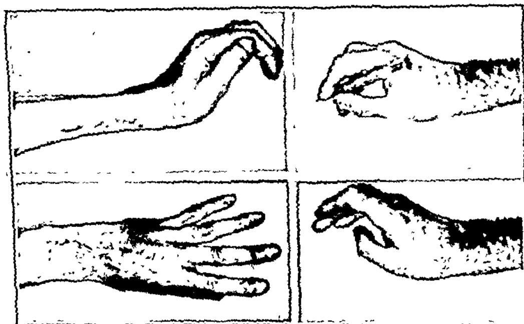


FIG 170 — The results after tendon transplantation in an irreparable radial nerve lesion.

IRREPARABLE NERVE INJURIES

We have encountered a small number of cases in which the injury was so extensive that an end-to-end nerve suture could not be performed and a nerve transplant has been impractical. In 2 of these patients, radial nerve lesions, we transplanted the pronator radii teres into the long and short radial extensors of the wrist to produce flexion of that joint. To restore extension to the fingers and thumb the flexor carpi ulnaris tendon was transplanted into the extensor tendons of the third, fourth, and fifth fingers; the flexor carpi radialis into the extensor pollicis longus and brevis, and the extensor tendons of the index finger. This was followed by a fairly successful resumption of function. (Fig 170.)

In another patient with a median nerve lesion, the tendon of the extensor ossis metacarpi pollicis was anastomosed *to the* flexor

pollicis longus and the extensor carpi radialis was joined to the tendons of the index finger. This provided flexion to the index finger and flexion and apposition to the thumb. In several instances it has been found advantageous to perform an arthrodesis on the thumb and fix it into a position of apposition and abduction.

We believe that tendon transplantations or joint immobilization are preferable to attempts to neurotize paralyzed muscles by uninjured adjacent nerves. Before a tendon is selected for transplantation, however, the ability of its muscle belly to perform the required task must be determined. A small weak muscle cannot perform the action of a stronger muscle though one may reasonably expect it to hypertrophy to some degree comparable to its added function. In addition, the synergistic action of muscles must be kept in mind. Each contracting muscle group has its antagonistic muscles which relax simultaneously. On the other hand, muscles which may appear to be anatomically antagonistic may contract simultaneously to fix a joint during all or part of the movement and thus are physiologically supplementary. Since mobility is the chief aim to be accomplished in the hand, it is requisite that the joints upon which the transplanted tendon is to act should not be restricted in their range of motion.

SIGNS OF RECOVERY OF FUNCTION

In reading the voluminous literature upon the results of peripheral nerve operations, one is struck by the numerous instances in which successful recovery of function is reported before histological regeneration could possibly have taken place. These erroneous reports, in our opinion, are due to a lack of appreciation of the fact that supplementary muscle movements may be developed by the patient very quickly, and secondly to overlap of sensory function by adjacent uninjured nerves. It is rare, indeed, that a true return of either motor or sensory function occurs before a period of six months following a nerve suture.

We have noted several points which are of interest in following the recovery of motor function and sensation. For example, the pronator and palmar muscles regain their activity first in the recovery of function following a *median nerve* lesion; then the flexors of the middle finger; and after that the flexor of the thumb. Flexion of the index finger and apposition of the thumb are impaired for a long period. This order of recovery of function is alike in complete and partial lesions. The complete recovery of motor function may be tested, as suggested by Pitres,[1] by having the patient scratch the

[1] Pitres, A La valeur des signes cliniques permettant de recommaitre dans les blessures des nerfs périphériques. A. La section complete d'un nerf. B. La restauration fonctionnelle, Rev. neurol., Paris, 23, 477, 1916

table with his index finger with the palm lying flat upon the table.

Recovery of sensation usually begins before that of motion, but it remains defective for a long period, particularly in the index finger. The presence of protopathic sensibility in the area of the median nerve has many times been interpreted as recovery of sensation. After true sensory regeneration occurs, the paresthesias and over-response to stimuli diminish progressively. This protopathic response is due to the overlap of fibers from adjacent uninjured nerves. When deep pressure is exerted over the area of the isolated supply of the median, that is, the distal phalanges of the index and middle fingers, diffuse pain may be elicited during regeneration. The return of sensation to one or another type of stimuli in the isolated supply of the median nerve is a true indication of sensory recovery.

In *radial nerve* lesions, the common extensor muscle is restored after the extensors of the wrist. Usually the extensors of the middle, ring, and little fingers show signs of recovery first although in some of our cases the index and middle fingers recovered their movement first. The extensors of the thumb and the abductor of the thumb regain their power last of all. Pitres has suggested two tests to determine the completeness of recovery in radial nerve paralysis. The first test consists in placing the arm in the attitude of a man taking an oath with the wrist and fingers extended, the thumb raised and separated from the fingers with the tendons of the extensors and long abductor of the thumb marking out the anatomical snuff box. Then the patient is requested to supinate his hand. The second test consists in having the patient place his little finger on the seam of his trousers with his fingers well extended and with the palm turned to the front.

In the radial, as in other nerves, interlacing of the borders of the loss of sensation to heat, cold, pain and touch, and recovery of tactile sensibility, particularly in islands away from the border of the former loss, constitute evidence of sensory recovery. The recovery of sensation to pin prick in this particular instance, we believe, cannot be looked upon as an important sign of recovery because of the very extensive overlap of the median and ulnar nerves.

In our cases, function in the small hand muscles recovered very slowly after *ulnar nerve* lesions, and recovery of the flexor carpi ulnaris and flexor digitorum profundus muscles always occurred first. Similarly, the adductor of the thumb recovers more frequently than the abductor of the little finger. Complete motor recovery may be shown by having the patient place the hand with the palm flat on the table and the fingers abducted. If the patient can then move his little finger inward and outward and scratch the top of

the table with the nail of that finger without moving his wrist, recovery of function may be said to have taken place. In many instances, motor recovery may begin without any evidence of sensory recovery in ulnar nerve lesions.

Return of function in the muscles supplied by the tibial portion of the *sciatic nerve* usually occurs earlier than in those innervated by the peroneal. Many sciatic nerve lesions which recover without operation may show good motor function without a corresponding improvement in sensation. In our peroneal nerve lesions which were operated upon, all have shown signs of motor recovery which began in the tibialis anticus muscle. In each instance the recovery of sensation has been slower, and the areas of recovery to pin prick stimuli have been very patchy.

RESULTS OF PERIPHERAL NERVE OPERATIONS

Many factors must be considered in any attempt to judge the results obtained in a group of cases such as these. The patient may be anxious to show a satisfactory result, or on the other hand, because of industrial insurance compensation, he may minimize the degree of recovery. Although there are exceptions, the interval of time which has elapsed between the injury and the time of operation plays an important rôle in the recovery of function. Particularly is this true if the muscles and joints have been neglected. Again, a previous infection may have been followed by extensive scar tissue damage. Finally, the persistence with which physical therapy, exercise, and use of the limb have been carried out affects the functional result tremendously. All of these factors are difficult to compile in any statement or classification of results.

The return of a patient to work cannot be used as a general index of recovery in nerve lesions even though stenography or typewriting might be successfully accomplished following the repair of an ulnar nerve lesion. If supplementary motility is not carefully excluded, many cases may be grouped as partially recovered. Likewise, the changes in sensory loss due to the overlap of adjacent uninjured nerves must be evaluated carefully. Changes in the color or nutrition of the skin are valueless as indications of recovery. The length of time following operation must be the same in comparing the results of any group of cases, and that the level of the lesions plays an important rôle in the recovery of function after nerve suture has become widely accepted.

In a study of the statistics of recovery of function in individual nerves there is a fairly common agreement that following end-to-end suture recovery is more complete in lesions of the radial nerve and

that the poorest results in the return of function are found after the repair of ulnar nerve lesions. The results in median nerve lesions lie midway between.

The level of the nerve lesion from the distal extremity of the limb would seem to be an important factor in determining the time of recovery. Foerster found evidences of recovery in median and ulnar nerve lesions in the lower third of the forearm at the end of three months as compared to twelve months when the lesion was in the axilla. This same relative time interval was also true in radial nerve lesions. Platt has stated that after suture of the radial nerve in the upper middle, and lower thirds of the arm, a return of muscle power was manifested in the supinator longus muscle at about the seventh, sixth, and fourth months, respectively.

As a result of the study of nearly a thousand peripheral nerve injuries received during World War I, 258 patients observed in civilian practice and 300 patients injured in World War II and examined carefully postoperatively, we have established certain criteria of the return of function after operations upon peripheral nerve injuries.

In the case of each individual nerve, the return of all forms of sensation in the isolated sensory supply of that nerve has been used as the basis for assuming a complete recovery of sensory function. Any criterion less than that may be logically explained on the basis of overlap from adjacent uninjured nerves and is therefore valueless. Improvement of sensory function is based upon a partial return of sensation in that area of isolated sensory supply.

We have noted that when sensation begins to return, the patient responds to pin prick in the area of isolated supply by withdrawing the part quickly in a defensive movement as if the stimulus were unusually painful. The patient explains that the sensation is very disagreeable, that it radiates, and that its after-effect is far out of proportion to the character of the stimulus. As this response to a pin prick becomes more nearly normal, we have noted that then the patient begins first to recognize a stimulus with cotton wool, and from that moment on the sensation to pin prick gradually becomes more and more normal. It is also true that this same ill-defined, spreading response to pin prick stimulation occurs in areas innervated by the sutured nerve other than in the isolated supply, but the overlap from adjacent uninjured nerves is responsible for this early return of sensation. This question of overlap of sensation is an extremely important one, and unless reports of the return of sensory function after suture or neurolysis of a nerve are restricted to the area of isolated sensory supply of that nerve, they are absolutely valueless.

In the determination of return of muscle function, the results obtained after each operation have been based upon specific motor movements which are the sole function of the muscles supplied by that individual nerve. Thus, muscle movements which might produce a more or less functioning extremity but which are trick movements employing uninjured adjacent muscles, must be disregarded. To state that an individual can use his extremity in certain occupations is an untrustworthy method of judging the results of a nerve operation.

The ability of the patient to abduct and adduct the middle finger with the palm of the hand flat on the table; to scratch the table with the tip of the little finger with the palm flat; to appose the thumb and little finger to make an "O"; the disappearance of muscle atrophy; and the proximity of the little to the ring finger have been used as the criteria of our results after ulnar nerve operations. Likewise, the ability of the patient to make a perfect fist; to scratch the table with the tip of the index finger if the palm of the hand is flat; and the disappearance of muscle atrophy are the signs of recovery of muscle function after median nerve operations. Following radial nerve operations, the return of motor function is indicated by the ability to dorsiflex the wrist with the fingers extended; to extend the fingers with the wrist flat; to pronate and supinate the wrist with the extremity upraised in the position of taking an oath; and to place the tip of the little finger on the seam of the trousers with the fingers and thumb extended, have been used as the indications of complete return of function.

The improvement of trophic disturbances has been judged by the healing of ulcers, the disappearance of glossy skin, and a return of normal color, and the disappearance of transverse and longitudinal ridges in the nails. Repeated electro-diagnostic examinations and electromyographic tracings now provide excellent purely objective methods of following nerve regeneration and the return of function.

Similar attention must be paid to the isolated sensory supply and supplementary movements in judging the return of function after injuries of the nerves of the lower extremity where impairment of function is not so disabling and where splinting devices are more efficient.

Very few of our patients were operated upon by us at the time of the original injury, which is undoubtedly the time for the most favorable result This was well illustrated in a six-year old child who severed the ulnar nerve at the wrist by falling upon a broken milk bottle The wound was scrubbed carefully with soap and water, the nerve ends were approximated accurately and the muscle repaired within four hours after the receipt of the injury. The early

return of motor and sensory function in the child was startling, and by the use of physical therapy measures a large degree of muscle atrophy was avoided. There is no doubt in our minds that if at the time of the injury a careful and painstaking approximation of the divided nerve ends was made, the end results of peripheral nerve lesions would be immeasurably improved. Too often a laceration is hurriedly repaired in unfavorable surroundings, when the patient should have been taken to an operating room and under anesthesia the divided structures should have been meticulously repaired.

This is not to say, however, that divided nerve trunks cannot be apposed with excellent results many months after the original injury. We have operated upon patients as long as two years following division of the nerve, but as has been emphasized, under such conditions the neuromas on the ends of the nerve are large; and the resection necessary to disclose normal nerve fibers in the central segment and a trunk free from scar tissue in the distal segment leaves a large gap in continuity which must be bridged. The most important factor, though, is the stiffened joints and fibrous muscles which follow improper splinting or complete neglect of these important effector mechanisms Regardless of how anatomically perfect regeneration of the nerve may be, the end result will be poor unless joints are kept mobile and muscles in tone by massage, passive movements, electrical stimulation, re-educative exercises and proper splinting.

The fact that the percentage of failures was far greater when nerve suture was performed at the time of the initial debridement than when it was deferred until after delayed wound closure in World War II injuries, does not necessarily mean that this dictum should apply to the early care of nerve injuries in civilian practice. There are many factors in military surgical procedures which mitigate against a properly and meticulously performed nerve suture at the time of the injury. Under the stress of warfare, opposing the nerve ends, or protecting the divided nerve ends providing for easy identification later, is often the best procedure. Failures in a delayed nerve suture are usually traceable to poor surgical technique and inadequate mobilization of the injured nerve.

The earlier repair of peripheral nerve injuries, the intensive use of physical therapy pre- and postoperatively, and the recognition of the true symptoms of recovery of motion and sensation will materially improve the end results of peripheral nerve injuries.

CHAPTER X

SURGICAL TREATMENT OF INTRACTABLE PAIN

PAIN is a symptom and should never be treated directly by surgical procedures unless the primary source of the pain cannot be removed. The surgical procedures employed for the relief of intractable (unmanageable) pain must be based upon proved anatomical and physiological facts. Because the nervous system is anatomically so organized that functional units are discretely placed, the accurate surgical application of a knowledge of physiology and anatomy may be used for the control of pain by interrupting those neural pathways which conduct certain sensory impulses from the source of irritation to the location of conscious interpretation within the brain. The record of the experimental work upon the physiology of pain performed by so many investigators throughout the years provides an interesting story, and the application of these experiments to the relief of pain has illustrated vividly the correlation possible between the laboratory and the surgical clinic However, in many instances, surgical empiricism has preceded known anatomical and physiological facts in attempts to relieve the unmanageable pain of many diseases.

The nerve fibers which carry sensory impulses from somatic areas come to the spinal cord in the peripheral nerve trunk and then separate to become concentrated in the posterior spinal root. As the posterior spinal root enters the spinal cord, the fine myelinated and unmyelinated fibers which convey pain and temperature impulses pass through the gray matter and ascend to the thalamus in the lateral spinothalamic tract of the opposite side. This tract lies superficially in the lateral column of the spinal cord anterior to the insertion of the dentate ligament. Its anterior boundary is the emerging fibers of the anterior spinal roots.

Given an area of the body the source of intractable pain, such as cancer metastases to the spinal column, it would be possible anatomically and physiologically to interrupt the fibers which convey pain by sectioning the peripheral nerve, the posterior spinal root, the lateral spinothalamic tract in the opposite side of the spinal cord or in the medulla, producing a destructive lesion in the thalamus, removing that part of the cerebral cortex in which pain is interpreted, or finally, by a lesion of a part of the cerebrum which makes the patient oblivious to pain.

The condition would rarely arise in which section of one peripheral nerve would stop pain from a given area and, if this were true, one would hesitate to sacrifice the motor components of the peripheral nerve when the sensory fibers alone could be interrupted in other locations.

Rhizotomy consists of the intraspinal section of the posterior spinal root between the spinal cord and the posterior spinal ganglion where the fibers cannot regenerate because of an absence of neurolemmal sheaths. In such an operation, therefore, one may by selection section the sensory components of the peripheral nerves to any selected number of dermatomes without interfering with the motor fibers. However, this procedure should never be done to the extent that an entire extremity is deafferented. Because of the anatomical and physiological overlap of the sensory supply from dermatome to dermatome, it is always necessary to section at least two posterior spinal roots above and below the area in which the pain is localized by the patient. In the case of intractable pain about the neck, shoulder and face, as is sometimes present in cancer in those regions, sensory rhizotomy of the upper cervical roots on the involved side, together with section of the sensory root of the trigeminal nerve, will give complete and lasting relief. Rhizotomy is particularly useful for the relief of intractable pain of a somatic nature involving the neck and trunk.

Although posterior rhizotomy of the upper thoracic roots for angina pectoris was first proposed by Danielopolu in 1923, we performed this operation first in 1933 with complete relief in the patient after section of 6 thoracic posterior spinal roots. Several others have corroborated the fact that angina pectoris can be successfully relieved by posterior root section of a sufficient number of segments. The disadvantages to the operation are that it requires a laminectomy and a bilateral section of the posterior spinal roots. However, it attacks the afferent pathways from the origin of the pain and its referred areas according to established anatomical and physiological facts.

Cordotomy consists of an antero-lateral section of the spinal cord and was first suggested by Spiller and carried out by Martin. An incision which begins at the level of the dentate ligament, anterior to its insertion, and finishes in the anterior spinal nerve rootlets, and is a minimum of 3 millimeters in depth, will section the fibers in the lateral spinothalamic tracts. This incision should be made bilaterally and at least 5 to 6 segments in the spinal cord above the highest level of pain.

This operation does not destroy the all-important proprioceptive senses, as does rhizotomy, and it may be applied for the control of

pain high in the thorax as might be produced by a carcinoma of the lung. In such instances, the cordotomy must be performed high in the cervical cord but becomes a rather dangerous procedure because of the origin of the phrenic nerves from the cervical 3, 4 and 5 segments of the cord. The inadequacy of a cervical cordotomy has led to the employment of incisions into the spinothalamic tract at higher levels in the medulla as advocated by Schwartz and O'Leary,[1] and at the junction of the pons and mesencephalon as was first done by Dogliotti[2] and later by others. Although sections made at these levels can give adequate relief of pain, the technical surgical factors involved make these procedures difficult and there may be secondary disabling symptoms due to the injury of adjacent structures.

Cordotomy is the operation of choice in patients with intractable pain which arises from abdominal or pelvic cancer, malignant tumors of the spine, sacrum or lower extremities, but it may also be used for pain of visceral origin. Experimentally, we have shown that painful impulses from the viscera, transmitted by the thoracic sympathetic trunk, are conducted upward by relays of short spinal paths with synapses in the gray matter of the spinal cord and do not pass into *the lateral spinothalamic fiber tracts in the white matter which serve* to transmit somatic painful impulses. The results of cordotomy operations performed for the relief of such visceral pains as tabetic *crises have uniformly been unsuccessful if the incisions have been* made to affect only the spinothalamic tracts. However, when such incisions have been carried deeper so that a portion of the gray matter has been included, relief from visceral pain may be obtained. Foerster demonstrated the microscopic sections of a spinal cord which showed the level of a cordotomy which had been performed with success for the relief of gastric crises. His sections showed unmistakably the extent of the lesion into the gray matter of the cord This is exactly what one would expect from the experimental facts.

It may be that pain comes into consciousness in the thalamus and not the cortex of the brain, the cortex serving only to regulate it in degree. Pain is a primitive sensation and logically it should have no accurate representation in the cerebral cortex, which is phylogenetically a new structure. *Ablation* of the *sensory cortex* has been performed for the control of phantom limb pain, and the reports of the results vary from excellent to poor with only temporary relief. There is a large *functional element in the pain of a phantom limb and* the results of surgical treatment for its control are difficult to deter-

[1] Schwartz, H G, and O'Leary, J L "Section of Spinothalamic Tract in Medulla with *Observations on Pathways for Pain*," Surgery, 9, 183, 1941

[2] Dogliotti, N "First Surgical Section in Man of the Lemniscus Lateralis (Pain and Temperature Path) at Brain Stem, For the Treatment of Diffuse Rebellious Pain," Anesth and Analg, 17, 143, 1938

mine no matter what type of operation may be employed. In this operation, performed under local anesthesia, the post-central gyrus and the adjacent parietal lobe area is exposed, and the cortex is explored by electrical stimulation until the conscious patient complains of reproduction or an increase in his pain. The gray matter for the corresponding limb is resected by a sub-pial dissection. Three of the five patients in our own series so treated had immediate and lasting relief from their phantom limb. This procedure has its disadvantages. A craniotomy must be done, sufficient cortex must be removed and Jacksonian convulsions may occur later.

There are some patients who complain of severe unmanageable pain for whom one of these already mentioned operations might not be considered as a proper procedure. Some patients have become deeply addicted to drugs, or there is an extreme state of tension, concern, and anxiety with the pain which require one of several methods of surgical attack upon the antero-medial nucleus of the thalamus. Such *thalamic destruction* may be carried out unilaterally or bilaterally, and many excellent results have been obtained with the unilateral operation. An elaboration of the technique of thalamic destruction has been made through the use of a modified sterotaxic instrument, originally designed by Horsley and Clarke for use in animals. When applied to the human head, it may be used to produce accurately placed electrolytic lesions within the antero-medial nucleus of the thalamus.

Operations upon the thalamus were first devised as a treatment in certain of the psychoses, the purpose of which was to overcome the fears and anxieties of the patient. The cell bodies of the fibers leading into the antero-medial nucleus of the thalamus lie in the cortex of the antero-medial portion of the frontal lobe, and by the operation of resection of such specific cortical areas, sometimes called "*topectomy*," the desired depressive effect upon the thalamus may be obtained. However, the same effect may be obtained by the equally effective, less complicated and less dangerous operation of *lobotomy*, or leucotomy, in which selected areas of the non-vascular white matter anterior to the lateral ventricle are severed under direct vision through trephine openings in the skull. Scarff[1] first performed a unilateral pre-frontal lobotomy for the relief of ipsilateral, contralateral, and bilateral pain. The advantages of a unilateral lobotomy over a bilateral lobotomy are, of course, that no notable personality changes are likely to occur.

The role of the *autonomic nervous system* in the physiology and anatomy of painful impulses has created experimental and clinical

[1] Scarff, J. E.: "Unilateral Pre-Frontal Lobotomy with Relief of Ipsilateral, Contralateral, and Bilateral Pain," Jour. Neuro. Surg., 5, 288, 1948.

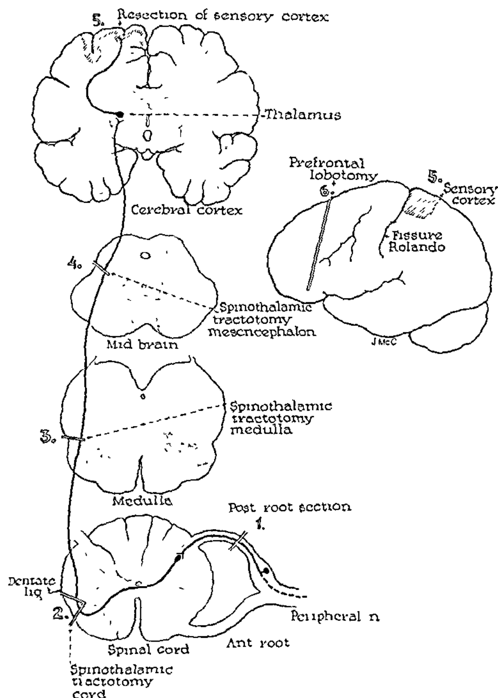


FIG. 171 — Diagram to illustrate several procedures used to interrupt pain pathways (1) Posterior rhizotomy, (2), (3), (4) Spinothalamic tractotomy in the spinal cord, medulla and mesencephalon, (5) Resection of sensory cortex, (6) Prefrontal lobotomy

interest for many years. It may be stated in the beginning that the anatomical evidence which exists that there are no afferent sensory pathways in the sympathetic system is fundamentally sound. It is only the efferent fibers of the autonomic system itself which are concerned with the production of pain and such afferent fibers as travel along with it belong to the ordinary spinal sensory system. Neuralgia, posterior-traumatic arthritis with osteoporosis, the diffuse aching or burning pain following frostbite, the cramping, vice-like pain in the calf muscles of patients with peripheral vascular disease can be relieved by excision of suitable portions of the autonomic nervous system. In all intractable cases of this kind, in which pain has become chronic and disabling, preliminary injection of procaine paravertebrally is an important prognostic procedure. To evaluate the role of sympathetic innervation in the pain, relief during the period of effective block of the sympathetic trunk with procaine must be complete. The persistence of relief for a number of hours indicates that perhaps repeated injections may result in further improvement with longer periods of freedom from pain and that ultimate recovery may be obtained without actual operation. When sympathetic block with procaine has given complete relief for a short interval, removal of suitable portions of the sympathetic chain and ganglia is reasonably certain to be successful. In the patient in whom prognostic block has been followed by no response, an operation upon the sympathetic ganglia and chain is not likely to succeed.

There are very fine small upper thoracic cardiac rami in addition to the well known cervical cardiac nerves, which run from the upper 3 or 4 thoracic sympathetic ganglia to the posterior cardiac plexus. Their physiological importance in pain conduction from the heart has been well established by the work of White, and they constitute an accessory pathway for the conduction of pain from the heart. It is White's opinion[1] that paravertebral alcohol injection of these thoracic sympathetic ganglia and their rami should be used for the poorest risk patients who suffer from cardiac pain and in whom direct surgical intervention, either by resection of the upper 3 thoracic sympathetic ganglia or by posterior rhizotomy, is contraindicated.

[1] White, J. C: "Technique of Paravertebral Alcohol Injection," Surg, Gyn., and Obs, 71, 334, 1940.

CHAPTER XI

SURGERY OF THE AUTONOMIC NERVOUS SYSTEM

At the beginning of this century knowledge of the anatomical relations and distribution of the autonomic nervous system far exceeded our understanding of its physiological functions. Much of this lack of investigative physiological facts was explained by the inherent difficulty of solving the problems of the autonomic system by experimental methods which employ animals and by the utter failure of clinical surgeons to employ the rigid control conditions of the experimental laboratory in evaluating the results of operations performed upon man.

An equally serious obstacle to the collection of accurate data from surgical procedures was the confusion in the minds of the clinician concerning the real meaning of the term "sympathetic nervous system." The neuro-anatomist had defined the autonomic system as a functional portion of the nervous system which supplies the glands, heart, and smooth musculature with their *efferent* innervation and divisible for convenience into cranial, thoracico-lumbar and sacral outflows. The neuro-physiologist found that in their response to certain drugs, like atropine and adrenalin, the cranial and sacral outflows agreed with each other and differed from the thoracico-lumbar outflow. Unfortunately, the name "sympathetic system" was applied to the latter portion of the autonomic system, and soon this term was *loosely and inaccurately used to designate the entire* autonomic nervous system.

In the last twenty years, the increasing abilities of neurological surgeons to modify visceral functions by interruption of the autonomic nerve pathways and their critical appraisal of the results obtained has made it possible to fill in the many gaps in our knowledge of the physiology of this important part of the nervous system, as well as to bring relief from several serious diseases.

Peripheral Vascular Diseases.—It has only been within recent years that *attempts have been made to bring order out of the* chaos which has existed in the terminology, pathology and classification of these diseases. A common error was to use a prominent symptom as the name of a disease, or to classify the arterial diseases according to small differences in the microscopic picture of sectioned vessels. As a result, the term "Raynaud's Disease" is still applied to various types of arterial disease because the outstanding

symptom may appear to be a blanching of the fingers or toes, or a case may be described as "erythromelalgia" because the feet become reddened when dependent, without any consideration of other evidence of arterial disease.

For practical purposes there are two important groups of peripheral vascular disease: (1) the organic obstructive diseases, and (2) the vasomotor imbalance conditions. The most common diseases in the first group are *thrombo-angiitis obliterans* and *arteriosclerosis obliterans*. In the second group, comparatively rare, belong the *vasospastic* syndromes such as Raynaud's disease and the *vasodilator* conditions characterized by erythromelalgia.

The vessels of the extremities are innervated by sympathetic fibers which originate from the sympathetic ganglia of the thoracicolumbar division of the autonomic nervous system. These fibers reach the vessels through the somatic nerves from which they are given off at regular segmental levels.[1] Histological investigations have demonstrated that the veins, capillaries, and arterioles are supplied by these sympathetic nerve fibers, and there is physiological evidence that stimulation of them produce vasoconstriction. They also exert a tonic influence over the peripheral vascular bed. Though vasodilator fibers are also present, they are not so readily demonstrable. Stimulation of sensory spinal nerves results in dilatation of the capillaries through the mechanism of an antidromic efferent conduction in those fibers.

A third nervous mechanism influences the peripheral vascular bed, and this is the local, or axon, reflex. The paths traveled in the axon reflex are generally regarded as at least in part sensory. Certainly the afferent path is sensory but two views are held concerning the efferent pathway. Bruce[2] has always held that it was sensory. Bardy[3] believes that the sensory collateral fiber forms a synapse around a local ganglion cell of the sympathetic system. The most conclusive evidence for a purely sensory spinal nerve pathway throughout is that an axonal reflex remains undisturbed after the sympathetic supply to the limb has been destroyed and that it

[1] This was demonstrated by the work of Kramer, J. G., and Todd, T. W. (The Distribution of Nerves to the Arteries of the Arm, With a Discussion of the Clinical Value of Results, Anat. Rec., 8, 243, 1914) and furnished a valid objection to [illegible]

[illegible] of higher centers.

[2] Bruce, A. N.: Ueber die Beziehung der sensiblen Nervenendi[illegible]

disappears from any part of the skin to which the sensory nerves have degenerated. Woollard's[1] clear description of the collateral branches of sensory fibers which shows that one and the same fiber may, by division, supply the arterial wall and special nerve endings, provides the anatomical proof for a purely sensory axonal reflex.

The experimental work of Hooker,[2] Krogh,[3] Rouget,[4] and others has shown that the capillaries are definitely capable of independent active alterations in their caliber from various stimuli. Stimulation of the sympathetic innervation produces an active capillary constriction which may occur independently of the arterioles. Stimulation of sensory spinal nerves results in a capillary dilatation through the mechanism of an antidromic efferent conduction in those fibers. Mechanical and chemical stimuli also affect the vascular bed. In a study of dermatographia Cotton, Slade and Lewis[5] have shown the effects of mechanical irritation of the skin which they attributed to capillary constriction or dilatation. The views of Krogh, and Hooker and Dale, are also in agreement that the capillaries are influenced by mechanical stimuli. In addition it has been shown that histamine, a chemical vasodilator, produces a marked capillary dilatation.

Attempts have been made to devise preliminary clinical tests which would give some guide to those patients who might be benefited by removal of the sympathetic supply to their extremities It is necessary to determine, first, whether the peripheral vascular disease is due to an organic occlusion of vessels; to a vasoconstrictor spasm, or to both; or is merely a peripheral symptom of a generalized disease.

The intravenous administration of typhoid vaccine with a subsequent rise in body and skin temperature in the ratio of 1 to 4 was suggested by Allen and Brown[6] as an accurate method of determining whether or not sympathectomy will be of benefit. Oscillometric and finger plethysmographic studies, the use of the salt solution absorption test, skin capillary reactions, the histamine flare reaction, the subcutaneous thermocouple needle, calorimetric observations, the introduction of a spinal anesthetic solution, capillary microscopy,

[1] Woollard, H. H.: The Innervation of Blood-vessels, Heart, 13, 319, 1926.
[2] Hooker, D. R. The Functional Activity of the Capillaries and Venules, Am. Jour. Physiol., 54, 30, 1920.
[3] Krogh, A.: The Anatomy and Physiology of Capillaries, New Haven, 1922.
[4] Rouget, C: Sur la contractilité des capillaries sanguins, Compt rend de l'Acad. d. sci., 88, 916, 1879
[5] Cotton, T. F., Slade, J. G., and Lewis, T. Observations Upon Dermatographism With Special Reference to the Contractile Power of Capillaries, Heart, 6, 227, 1917.
[6] Allen, E. V., and Brown, G. E Raynaud's Disease, A Critical Review of Minimal Requisites for Diagnosis, Am Jour Med Sci, 183, 187, 1932.

paravertebral sympathetic trunk block, and anesthetization of the peripheral nerves have also been employed. Coller and Maddock[1] have studied the skin temperature response of normal subjects to varying room temperatures and to high environmental temperatures. They have shown in applying the procedure to subjects with peripheral vascular disease that a failure to reach a normal vasodilatation level was due to the presence of organic vascular obstruction. The ease of application of this test of environmental response, without danger and with little inconvenience to the patient recommends it. All of these tests are a measure of the ability of the vessels to dilate and are not, as is often assumed, indicators of the degree of vasospasm present.

The greatest amount of work has been done with skin temperature studies, and unwarranted conclusions have been made from a failure to appreciate not only the functions of the skin but also the factors which influence the skin temperature. The temperature of the skin represents the sum total effect of the heat brought to it by the blood, the heat developed by metabolism in the surrounding tissues, the conduction and radiation of heat by those tissues, and the cooling effect of the evaporation of sweat and environmental changes. It may be suspected, therefore, that skin temperature is not an accurate index of the degree of peripheral blood flow.

Our experience with sympathectomy in the treatment of vascular disease is concerned with cases of thrombo-angiitis obliterans and Raynaud's disease. By the latter we mean that condition which tends to affect all digits symmetrically, has varying degrees of vasomotor disturbances, does not produce severe pain or gangrene except in the late stages, and occurs predominantly in females. The rationale for applying sympathectomy in cases of thrombo-angiitis obliterans rests on the fact that in many instances there is no complete obliteration of the main arteriole channels and that vasoconstrictive disturbances often occur to a sufficient degree in unoccluded and collateral vessels to simulate Raynaud's disease.

The story of our first patient with thrombo-angiitis obliterans upon whom we operated represents one of our most successful results following sympathectomy.

In 1925, a patient was seen who complained of a severe aching, burning pain in the sole of his right foot, particularly after walking. This had increased gradually in severity until he could walk only 4 or 5 blocks without severe discomfort and pain. As the condition progressed, he suffered pain while at rest. When his extremity was horizontal, his foot would become pale and he would complain of numbness and paresthesias. If the extremity was dependent, the foot became red and purple in color.

[1] Coller, F. A., and Maddock, W. G.: The Differentiation of Spastic From Organic Peripheral Vascular Occlusion by the Skin-temperature Response to High Environmental Temperature, Ann. Surg., 96, 719, 1932.

The dorsalis pedis and posterior tibial pulse in the right lower extremity were difficult to palpate in contrast to those in the left leg. A marked hyperesthesia was present over the dorsal surface of the foot. If the foot was kept warm with the extremity in the horizontal position, the toes and dorsal surface of the foot would keep their normal pink color. The nails of the second and fourth toes were brittle and longitudinally striated.

When examined a year following a lumbar sympathectomy, in which the right chain from the second to the fourth ganglia inclusive was removed, he had returned to his work as a tailor. He had had no recurrence of attacks of pain or pallor in the right foot. The surface temperature readings of the right foot, which had risen several degrees immediately after operation, had returned to the preoperative level.

It has been the experience of many surgeons that the patients with thrombo-angiitis obliterans so operated upon have obtained relief from the marked color changes which occurred upon exposure to heat or cold or to a change in posture and have been benefited greatly in the relief of their attacks of pain. Our experience has been that patients have obtained the greatest benefit in those instances in which their disability was minimal. Ulcers have healed, the condition of the nails has improved, and in other respects there has been a very definite indication of an improvement in the collateral circulation, which in some instances at least appears to have prevented the progression of gangrene.

The weakness in the argument in favor of sympathectomy for *thrombo-angiitis obliterans lies in the tendency to place too much* emphasis on the vasomotor element which may be present. It is only in the early stages of the disease that vasomotor disturbances are prominent.

However, Reid[1] has called attention to many of the simple but fundamental principles in the general care of peripheral vascular diseases which have been discarded or ignored in the enthusiasm which has developed for a new method of surgical treatment and which can be used with success in cases of thrombo-angiitis obliterans. The position of maximum benefit to the circulation with the extremities at rest; the harmful effect of cold; the use of baths, oils, and greases to make the skin as soft as possible; the avoidance of trauma; the elimination of foci of infection; the use of voluntary exercises; and the employment of the Pavaex apparatus to produce a passive hyperemia all should be emphasized to the patient. This is particularly true since the underlying cause of this condition is unknown and cannot be removed at present, at least, by a surgical procedure.

The syndrome commonly known as *Raynaud's Disease* has occupied an unusual place in medical literature. Nothing of importance has been added to Raynaud's original description, and it is

[1] Reid, M. R. *Diagnosis and Treatment of Peripheral Vascular Disease.* Am. Jour. Surg., 24, 11, 1934.

as true now as it was fifty years ago when Hutchinson emphasized the fact that "Raynaud's phenomena" was not a clinical entity at all but a group of symptoms occurring in many different conditions.

There is a wide difference of opinion regarding the nature and cause of the vascular phenomena observed in the attacks of Raynaud's phenomena, and this is reflected in the methods of treatment. Raynaud thought the spasms were due to an abnormal vasomotor tone, but in a very comprehensive discussion Thomas Lewis[1] concluded that the primary cause of spasm of the digital vessels is not an abnormal vasomotor tone but a local fault of the vessels. This conclusion was based in part upon observations which showed that the digital vessels, or even parts of them, are brought to complete contraction by cold, to which the vessels in these patients appear to be unduly sensitive. It was also based in part on the demonstration that release of vasomotor control by anesthetization of the peripheral nerves does not prevent the vessel so affected from still being brought into a condition of spasm by suitable local stimulation. However, these changes do not occur after section and degeneration of the peripheral nerve fibers. These are exactly the same conditions under which the "flare" reaction to histamine disappears, and for this reason Lewis attributed the local spasm to an axon reflex in the sensory cutaneous nerves.

Our first case was cited by Lewis to support his contention that spasms of the vessels could still be produced after removal of the sympathetic supply to the upper extremity.

The first patient was operated upon in 1926, and the entire cervical sympathetic chain and all of the ganglia from the superior to the stellate inclusive were removed. Ordinarily the patient's hands were slightly redder in color than those of the average individual. During a vascular spasm, which could be produced by immersing the hands in cold water or exposing them to cold air, the color of the fingers rapidly changed to a bluish-gray. This involved all of the fingers and thumb down to the metacarpophalangeal joints except the fifth fingers which became discolored only over the distal phalanges. During these attacks the radial pulses were full and strong, but the fingers felt cold and clammy. They were very tender to the slightest pressure, particularly over the extremities. The tips of the fingers and thumbs were hard and board-like and were very painful at all times but more so during an attack. The skin over these areas was scaly and indurated. Immediately after operation, there was a rise in the surface temperature of the right upper extremity, but this had returned to normal within a month. Two months later the hands were immersed in cold water. After fifteen minutes no change had occurred in either hand, but as soon as they were brought into the air the attack of syncope and asphyxia began The change in color to the typical chalky bluish-

[1] Lewis, Thomas: Experiments Relating to the Peripheral Mechanism Involved in Spasmodic Arrest of the Circulation in the Fingers, a Variety of Raynaud's Disease, Heart, 15, 9, 1929.

white blanching occurred quickly in both hands. The return to normal color of the right hand was completed five minutes before that in the left.

We did not draw the conclusion from the observations made upon this patient, namely, that sympathetic denervation of the limb failed to prevent cold from producing complete spasm in vessels of the fingers. Lewis' argument was criticized on the basis of an incomplete sympathetic denervation to the upper extremity in an attempt to overcome the theoretical difficulty of the assumption that Raynaud's disease is due to vasomotor spasm.

Our later cases, in which the stellate, second and third thoracic sympathetic ganglia were removed, were followed by the same results corroborated in these instances by plethysmographic tracings. Smithwick, Freeman, and White[1] also observed the recurrence of vascular spasm after complete sympathectomy of an extremity in patients with Raynaud's disease. They believe that this is due to sensitization of the sympathectomized extremities to adrenalin in the circulating blood stream. Smithwick[2] has stated that the upper extremity can be thoroughly sympathectomized by interrupting the outflow from the second and third dorsal segments and by dividing the sympathetic trunk below its third ganglion. By this operation he believes that the preganglionic fibers to the extremity are interrupted completely. He has stated that the immediate results of the operation have been uniformly good, but the later results vary because of regeneration of the sympathetic fibers. Therefore, he attempts to prevent this regeneration by ligating the distal end of the divided sympathetic trunk and covering it with a silk cylinder. He also removes a part of the anterior root from the second and third segments by separating the attachment of the arachnoid and then gently teasing the root out. Our own experience with this operation has not been any more satisfactory than removal of the stellate and second thoracic sympathetic ganglia. We have used the latter operation on one extremity and Smithwick's modification upon the other without being able to detect any differences whatever in the results

Johnson[3] has reported the results of a study of 22 patients with Raynaud's disease, 5 of whom we operated upon. He found that the relief of mental anxiety, the improvement of an anemia, and the

[1] Smithwick, R. H., Freeman, N. E., and White, J. C.: Effect of Epinephrine on the Sympathectomized Human Extremity, Arch Surg, 29, 759, 1934

[2] Smithwick, R H Modified Dorsal Sympathectomy for Vascular Spasm (Raynaud's Disease) of the Upper Extremity, Ann Surg, 10, 339, 1936, The Problem of Producing Complete and Lasting Sympathetic Denervation of the Upper Extremity by Preganglionic Section, ibid, 112, 1085, 1940

[3] Johnson C A: A Study of the Clinical Manifestations and the Results of Treatment of 22 Cases of Raynaud's Symptoms, Surg, Gynec. and Obst, 72, 889, 1941.

search for and treatment of an underlying specific disease gave relief to the majority of these patients, including some who had been previously operated upon unsuccessfully. He concludes that the vascular changes during a Raynaud's attack are not necessarily due to an active vascular constriction but may be the result of a vasodilatation in the palmar arch with a diversion of blood from the fingers and a passive collapse of the vessels.

One of our cases is particularly instructive because it illustrates the lack of our knowledge of the sympathetic nervous system in its relation to the pathogenesis of Raynaud's syndrome.

In 1930 all of the fingers of this white female, aged forty-seven years, turned white to the metacarpophalangeal joints and then soon became blue, red, and finally normal in color. These attacks continued and became more severe in their frequency and duration. During the winter time it became necessary for the patient to stay indoors practically all of the time and even then she would have two or three attacks each day. Cool air more often than water would precipitate an attack, and she could remember only one occasion when an attack was associated with an emotional upset.

The attacks were relieved by immersion of the hands in cold water followed by warm water. When she was eight years old, she had typhoid fever, and at thirty she had influenza.

Examination was negative except for disturbances of the growth of the finger nails, sclerodactylia, and stiffness of the finger joints.

The stellate and second thoracic sympathetic ganglia were removed on both sides in January of 1934, and for a time the patient thought she had improved, particularly as the warm weather set in. The following winter the symptoms returned as severely as before the operation, and ulcers appeared upon the tips of several of the fingers.

Although the patient had repeatedly denied a luetic infection, the blood Wassermann reaction was weakly positive, and on this evidence alone antiluetic treatment was started. This was followed by complete relief of the Raynaud's attacks although ulcers still occur on the finger tips. The scleroderma has definitely improved.

Studies of the peripheral circulation were made with the Johnson plethysmograph in the fall of 1935. There was an absence of pulsatile changes in the control [illegible]
particul [illegible]
block in [illegible]
ture in [illegible]
fourth and fifth fingers, but the skin temperatures decreased in the fourth and fifth fingers of the right hand which is the usual finding in normal individuals following this procedure.

In other words, in this patient median nerve block produced an improvement of the circulation in the fingers innervated by the median nerve. Presumably, the operation should have destroyed the vasomotor fibers travelling in the median nerve; and therefore, it is difficult to understand this result unless some vasomotor fibers still remained or somatic fibers may have a vasomotor function.

30

Since the patient does not perspire in the hands or over the upper extremity, it is not likely that regeneration of the vasomotor fibers would occur without regeneration of the fibers controlling the sweat glands.

In another patient, the stellate and second thoracic sympathetic ganglia were removed on one side and section of the preganglionic fibers and division of the sympathetic trunk below the third thoracic sympathetic ganglia was carried out on the other side without any difference whatever in the relief of symptoms and without any changes in the plethysmographic studies.

In other words, in our experience the local cause of the attacks apparently remains unchanged in spite of the type of operation carried out for the relief of Raynaud's attacks in the upper extremities. We agree with other neurological surgeons that the results of lumbar sympathectomy for the relief of Raynaud's attacks in the lower extremities are strikingly superior to those obtained in the upper extremities [1]

In summary then, one may say that there is an early relaxation of the peripheral vessels following sympathectomy. However, there is a progressive recovery of the independent tone of those vessels so that the circulation reaches its preoperative level after a comparatively short time. This would indicate that the sympathetic fibers exert a tonic constrictor control over smaller vessels. Local relaxation of blood-vessels to heat or constriction by the local application of cold is subject to little or no central control. Finally, it must be kept in mind that there are many variable factors concerned in the control of the peripheral circulation in which the vessel musculature and caliber are but a part.

The Autonomic Nervous System and Pain.—Our own opinions concerning the relation of the autonomic nervous system to the production and relief of *pain* has been explained in Chapter X.

Anatomical and physiological evidence has been presented to show that the autonomic nervous system has no direct relation to pain in the extremities. Sympathetic fibers carry only *efferent* impulses and influence pain sensation in an indirect manner by altering the threshold of impulses carried by *afferent* somatic sensory nerves. In spite of this proof the surgical literature contains many

[illegible] ganglionic section been its upper vasomotor [illegible] ganglia Therefore, postganglionic fibers in the sciatic nerve are left intact but are disconnected from the central nervous system. Smithwick attempts to produce an analogous situation in the upper extremity by sectioning rami carrying preganglionic fibers

clinical reports of evidence which tends to controvert these facts. Cases of causalgia and amputation stump neuralgia have not been relieved by section of posterior spinal roots or other logically indicated procedures. Instead of properly questioning the completeness of the operation performed, other pathways are assumed to exist and as a result it has been concluded that sympathetic fibers, or even anterior spinal roots, carry afferent painful impulses. This assumption of the proof of physiological facts by negative evidence has resulted in contradictory opinions which have further confused the practicability of performing surgical procedures.

Every surgeon has encountered the patient who, after an amputation, complains of severe burning pain in the entire extremity or in the amputation stump. Frequently, a dense neuroma develops in the divided nerve trunks which involves the adjacent artery, and then the patient suffers from a pain similar to causalgia. In such cases the neuroma should be resected and the nerve trunk injected with alcohol to produce a dense fibrotic scar free from nerve endings. At the same time the vessel must be dissected free and placed in an uninjured muscle plane to prevent recurrence. It has been our experience that no procedure directed toward the sympathetic system will relieve these pains; and as a matter of fact, they are unnecessary if proper surgical therapy is carried out in the beginning, or if in a certain group of patients, serious psychoneurotic factors are accurately recognized and treated.

We have had an experience which demonstrated the inadequacy of periarterial sympathectomy in a patient with causalgia.

In falling from a ladder, the patient's left arm struck the top of a picket fence, and one of the iron top spikes penetrated the medial aspect of the arm near the junction of its middle and upper thirds. There was slight oozing of blood from the wound, but no loss of function was noted and a slight pressure bandage sufficed. The next day the patient developed a shooting pain in the whole hand though there was no pain in the arm. This continued for two weeks but without any loss of function in the hand or arm. Then the pain became more severe, burning, constant, and limited to the radial half of the hand. The ring and middle fingers became stiff and the finger nails glossy. He noted that moisture lessened the pain and that he could bear it if the hand was kept damp. He could not bear to have the hand touched; a loud noise, a sudden jarring or anything dry touching his hand would increase the pain. The forearm was held flexed and the hand was flexed on the forearm with the fingers drawn together at the tips in the form of a cone. The nails were long and ridged. The skin was shiny, opalescent and seemed tightly drawn

At another hospital a periarterial sympathectomy was performed on the brachial artery above the site of the wound, which was indicated only by a small scar.

At operation, we exposed the median nerve at the site of injury. A fusiform swelling of the nerve was present at the junction of its middle and upp

thirds, and at this point it was surrounded with scar tissue, which in turn was firmly adherent to the brachial artery. The nerve seemed to pulsate with each beat of the vessel. The nerve was freed from the artery; the neuromatous portion excised; and an end-to-end suture performed with immediate and continued relief.

The relief of the pain of causalgia following peripheral nerve injuries by sympathectomy has been discussed in Chapter IX.

Epilepsy.—We have had no personal experiences with sympathectomy in the treatment of epilepsy, but White[1] in his excellent monograph has summarized his experiences with 17 patients. Three cases of severe epilepsy so operated upon have been free from attacks for periods of over three years In four other patients the severity of the attacks was diminished, and in the remaining 10 cases there has been no improvement of any kind. White has stated further, and this is in entire agreement with our own views, that if sympathectomy alone produced improvement, he knows of no method of selecting the cases in which such an operation may succeed. The surgical literature is full of the reports of entirely non-specific operations, such as colectomy for example, which have appeared to give a certain number of good results so that one must conclude that no final conclusions can be drawn.

At one time or another every neurological surgeon has observed a focal area of spasm in the cortical blood-vessels associated with a convulsive seizure. Stimulation of the motor cortex with the production of an attack produces an identical phenomenon. These observations do not necessarily lead to the conclusion that epilepsy is due to vasoconstriction in the cortex. Even if this were true, the reflexes need not involve the cervical sympathetic ganglia. As Penfield has stated, the vasospasm may be produced purely through the mechanism of a local vascular plexus

Migraine.—This proved ability of the cerebral arteries to constrict has led to the theory that *migraine*, with its attendant, transient hemianopsia and aphasia, is due to a vasospasm. The severe headache is assumed to be transmitted over afferent sympathetic fibers though none have ever been demonstrated to exist either by anatomical or physiological experiments It may be possible that sympathetic fibers, purely efferent as they are, are involved in migraine, particularly since unilateral lacrimation and sweating phenomena are observed, but at present the value of sympathectomy is unknown The problem remains an experimental and clinical one and should be treated as such to avoid premature conclusions from insufficient evidence or incorrect premises.

[1] White, J. C The Autonomic Nervous System, New York, The Macmillan Company, 1935.

Retinitis Pigmentosa.—After removal of the cervical and upper thoracic sympathetic ganglia, lasting dilatation of the retinal arteries has been reported. Consequently, it was natural to believe that sympathectomy would benefit patients with *retinitis pigmentosa*, a disease characterized by thread-like arteries, night blindness and "gun-barrel" fields of vision. Though Kerr[1] and others have reported improvement in a small number of patients in the early stages of the disease, it is the opinion of our associates in the ophthalmological department that sympathectomy has been of no benefit in 5 patients operated upon. Finally, Verhoeff's[2] pathological studies indicate that as the retinal vessels run outward from the optic disc, their adventitia increases in thickness so that the vessels may even consist of solid strands of hyaline connective tissue. The futility of a sympathectomy in the presence of such pronounced organic changes is obvious.

Megacolon.—The only condition in the gastro-intestinal tract benefited by sympathectomy is *megacolon*, if it be of neurogenic origin. Morton and Scott[3] have said that these cases can be differentiated by the onset of active peristalsis after the administration of spinal anesthesia. The physiological basis for removal of the sympathetic supply to the large, dilated bowel is well established. Stimulation of the thoracico-lumbar sympathetic nerves produces contraction of the rectal sphincter and relaxation of the bowel above. Paralysis of these nerves leaves the sacral autonomic outflow in control; the sphincter relaxes, and tone in the bowel musculature is increased.

The postoperative course of these cases shows a striking improvement which can be maintained by the careful and persistent co-operation of an attentive patient. This has been the case with a patient we have operated upon. Unfortunately, many of these young patients are feeble-minded, and the final result often falls short of what might logically be expected.

Cord Bladder.—There exists a similar antagonistic innervation of the urinary bladder by the sacral and thoracico-lumbar autonomic outflows. Stimulation of the sacral fibers causes the internal vesical sphincter to relax and the detrusor muscle to contract. Contrariwise, the thoracico-lumbar fibers cause the internal vesical sphincter to contract, thus micturition is inhibited and bladder filling is favored.[4]

[1] Kerr, H H The Surgical Treatment of Retinitis Pigmentosa, Am. Jour. Surg., 38, 364, 1935.

[2] Verhoeff, F. H : Microscopic Observations in a Case of Retinitis Pigmentosa, Arch. Ophthalmol., 5, 392, 1931.

[3] Morton, J. J., and Scott, W. J. M . The Measurement of Sympathetic Vasoconstrictor Activity in the Lower Extremities, Jour. Clin. Invest , 9, 235, 1930.

[4] Learmonth's clinical experiments furnished direct proof that sympathetic fibers controlled the region of the vesical trigone and the internal sphincter.

As a result of spinal cord injuries, myelitis, tabes dorsalis, or other lesions, this physiological balance may be disrupted and the sympathetic innervation holds full sway uninhibited. Learmonth[1,2] suggested resection of the superior hypogastric plexus at the sacral promontory (presacral nerve) to correct this imbalance. Excellent results follow in properly selected cases in which examination points definitely to involvement of the sacral portion of the spinal cord. Since lesions in the cord above the lumbar segments involve the thoracico-lumbar outflow, no advantage can come from the operation under such circumstances. We have observed a marked improvement following section of the presacral nerve in a child with a cord bladder, the result of a spina bifida.

Resection of the presacral nerve for the relief of intractable pain in the bladder and prostate has a limited value; whereas, cordotomy is at once a more effective and less dangerous operation as we have had occasion to prove to our own satisfaction.

Dysmenorrhea.—There have been many reports in the literature of the favorable results obtained by division of the lumbar sympathetic chains and resection of the superior hypogastric plexus for the relief of *dysmenorrhea* and the intractable pain associated with advanced malignant uterine disease Here again, good surgical judgment is required in choosing between non-operative or surgical gynecological procedures, cordotomy, and sympathectomy.

Spastic Paralysis.—Though sporadic reports in the literature upon the results of operations upon the autonomic nervous system had been made by Jaboulay, Leriche, Jonnesco and others, it was not until the combined reports of Hunter and Royle appeared upon the relation of the "sympathetic" system to *muscle tone* that the present interest of surgeons in the autonomic nervous system began.

Assuming as proved the principle that all striated muscle has a dual innervation from the cerebrospinal and autonomic nervous systems, these investigators predicated that increased muscle tonus, due to various types of lesions, could be decreased by interruption of the innervation of skeletal muscles from the thoracico-lumbar outflow or "sympathetic" system. Royle based his operation of cutting the sympathetic rami to the brachial and lumbosacral plexuses on three erroneous anatomical and physiological conceptions. (1) that there existed "contractile" and "plastic" muscle tone, the latter being increased in patients with a spastic paralysis; (2) that "plastic" tone was a function of the primitive red muscle fibers which were

[1] Learmonth, J R The Treatment of Certain Types of Vesical Paralysis. Jour Urol, 26, 229, 1931.

[2] Learmonth, J R and Braasch, W. F. Resection of the Presacral Nerve in the Treatment of Cord Bladder, Surg, Gynec and Obst., 51, 494, 1930

innervated by sympathetic neurones, and (3) that the extensor rigidity present in a decerebrate animal, which is the best example of experimentally produced increased muscle tone, could be decreased by sympathectomy.

It seemed obvious to us[1] that several pertinent questions immediately suggested themselves for answer. Was there definite evidence in favor of the dual innervation of striated muscle? What effect does sympathectomy have on increased muscle tone? What are the various clinical types of increased muscle tone? What mechanisms in the central nervous system control muscle tone? What part do these mechanisms play in the production of abnormal muscle tone? How far and with what degree of success could these theories be applied to given clinical cases of muscular hypertonicity?

Prior to 1928, there was evidence of a controvertible nature, which rested mainly on Boeke's observations, that there was a dual innervation of skeletal muscles by the cerebrospinal and autonomic systems, but work by several investigators since then has utterly failed to corroborate Boeke's work. It is rather generally accepted at this time that every ending seen on a skeletal muscle fiber, whether non-myelinated or myelinated, originates from the cerebrospinal system and that any sympathetic neurones found in striated muscle serve only to innervate its blood-vessels.

Our own experiments on animals soon convinced us that removal of the thoracico-lumbar ganglionated trunks in cats produced no effect on normal muscle tone which could be observed or recorded and that the onset and maintenance of decerebrate rigidity in cats was unchanged after the removal of the sympathetic innervation to an extremity. Likewise, there has never been any proof of the converse experiment that sympathetic stimulation causes an increase in muscle tone. With the exception of Royle's experiments on goats, all of the experiments on mammalians have been in accord, and the weak point in Royle's evidence was his statement that a number of days had to pass after sympathectomy before the disappearance of "plastic" tone.

Our own evidence obtained from performing the operation upon patients with Parkinson's disease, Little's disease, and degenerations of the spinal cord, all of whom were chosen for operation by Royle, was quite negative. The follow-up examination of these patients several years later confirmed our original opinions. The late results in a group of patients operated upon by Royle in England were disheartening, and the conclusion was reached by impartial observers that "The operation therefore appears to have no place of value in

[1] Kanavel, A. B., Pollock, L. J., and Davis, L.: Relation of the Sympathetic Nervous System to Muscle Tone, Arch. Neurol. and Psychiat., 13, 197, 1925.

the treatment of spastic weakness." As we suggested in 1925, the autonomic nervous system may have some function dealing with the metabolism of muscle so that under certain conditions the contractility of a muscle may be changed by removal of sympathetic impulses. But, as White has said, "The secondary benefits derived through increasing the cutaneous circulation and altered muscular metabolism do not justify the severity of the operation." Particularly is this true when it is realized that the operation rests on inadequate physiological facts based solely upon an inaccurate interpretation of experimental and clinical evidence.

Essential Hypertension.—An accurate evaluation of the results of surgical intervention in the treatment of essential hypertension cannot be made unless one has a well defined concept of the multiplicity of factors which are involved in this clinical entity. Our knowledge of hypertension may be divided into four historical periods The first began with Richard Bright (Circa 1827-1836). He associated the cardiac hypertrophy, which was observed in patients with renal disease, with changes in the blood which he believed capable of increasing the work of the heart, or affecting more vessels in such a manner as to create an obstruction to the passage of blood through the different subdivisions of the vascular tree

The second period began in 1880 with the introduction by vonBasch of the sphygmomanometer. Very rapidly thereafter Albutt, Suchard, Janeway, and Volhard and Fahar, made their separate contributions. The third and rather recent period of our knowledge of hypertension may be dated by Goldblatt's work in 1934, when he succeeded in reproducing hypertension experimentally by partial occlusion of the renal arteries. The fourth, and presently influential theory of the general adapatation syndrome and the diseases of adaptation, including hypertension, may be traced back to the establishment of the broad general concept of vasomotor tone and balance by Anrep, and Cannon's principles of homeostasis.

This disease, the prealbuminuric stage of chronic Bright's disease of Mahomed, the latent arteriosclerosis of vonBasch, the hyperpiesia of Albutt, the presclerosis of Suchard, the hypertensive cardiovascular disease of Janeway, the benign and malignant sclerosis of Volhard and Fahar, is most widely known in the United States as essential hypertension.

By general agreement, essential hypertension includes those patients with chronic hypertension which neither clinically nor structurally can be shown to have resulted from an antecedent inflammatory disease of the kidneys or from obstruction to the urinary tract. The kidney is denied a role in the production of essential hypertension, rather there is more probability than the possibility

that sclerotic narrowing renal arterioles is a consequence of the elevation of blood pressure. All that the term, essential hypertension, really means is non-nephritic hypertension and, of course, includes many conditions having the common characteristics of an elevation of blood pressure with the equally common negative characteristic of the absence of primary renal disease. As Fishberg has said, "This definition is seriously defective, in that it defines solely by exclusion but in our present state of ignorance of the actual etiology, it is neither practical nor feasable to define it in any other way. The very term—essential hypertension—is a confession of our ignorance and this, of course, is its chief virtue."

Pathogenesis.—The pathogenesis of hypertension involves the autonomic nervous system, kidneys, liver, endocrine glands, and blood vessels.

In some individuals, emotional tension builds up as a result of conflict between the individual and his environment and is discharged by way of the hypothalamus and sympathetic nervous system. General constriction of the arterioles of the body, and in particular of the kidneys, results from this sympathetic system stimulation. Increased resistance to blood flow results and a hypertension follows which is short-lived. As a result of the arteriole constriction in the kidneys, a relative ischemia and anoxia result. According to one theory, it is this ischemia which acts as a trigger to cause the elaboration of a pressor substance, or substances, by the kidney. The release of this substance together with adrenal hormones results in a further and more sustained vasoconstriction. At this stage, the blood pressure is labile and may return to normal after the process has run its course. Repeated episodes of this kind result in organic changes both in arterioles of the kidney where a permanent relative ischemia with continued production of pressor substances occurs and in other arterioles situated generally throughout the body. The higher the blood pressure rises, the greater the degree of the resulting arteriole sclerosis and the greater the degree of arteriole sclerosis, the higher is the resulting blood pressure which causes even more sclerosis and thus a vicious cycle is created.

Some sixty pressor substances have been implicated in the pathogenesis of essential hypertension and about one-half of these are thought to be formed in the kidney. Other organs implicated are the adrenals, liver and brain although it is possible that other tissues of the body with this function have not as yet been implicated.

Goldblatt[1] demonstrated that hypertension could be produced

[1] Goldblatt, H., Lynch, J., Han[illegible] [illegible] Studies on Experimental Hypertension; [illegible] ystolic Blood Pressure by Means of Ren[illegible] 1934.

and maintained for months or years in animals in which a clamp was applied to the renal arteries which would result in a reduction of the blood supply to the kidneys. Later Page[1] stated that the renal ischemia and anoxis thus produced, caused the elaboration of renin, *an enzyme, from the kidney which activated hypertensinogen, an* alpha globulin, found in plasma and produced by the liver, to form hypertensin which caused arteriole constriction.

Zweifach and Shorr[2] believed that any concept of hypertension *must include not only the arterioles but the capillary bed as well.* These capillary vessels have contractile properties of their own, and are an important factor in controlling hydrostatic and osmotic pressure.

These investigators have described a vasoexcitor material elaborated by the kidney but distinct as a new entity and separate from hypertensin. The function of this substance, VEM, apparently, is to potentiate the smooth muscle of the arterioles and capillaries in such a way *as to cause more intense constriction then would ordinarily* occur in response to a vasoconstrictor stimulus. They further predicate that VEM may be counteracted by a vasodepressor material VDM, formed in the liver.

The adrenal glands take part in raising the blood pressure in response to stress situations. An abnormal production of adrenal hormones may be of pathogenic significance in the production of hypertension. The stimulus to the adrenal medulla may be mediated from the brain over the hypothalamic-pituitary pathway with utilization of the adrenotropic hormone and also directly by the sympathetic nervous system which innervates the adrenal medulla.

That the renin-hypertensinogen-angiotonin theory of hypertension was not satisfactory, unless the adrenal cortical effect was added, has been recognized. Page has made this theory tenable by including the action of desoxycorticosterone acetate which is elaborated in the adrenal cortex primarily in the outer zone (zona glomerulosa) This theory would then state that renal ischemia acts to produce renin which activates hypertensinogen to form hypertensin (angiotonin) which in turn acts, in addition to causing arteriolar constriction, to increase the elimination of sodium by the kidney. This in turn calls in the adrenal cortex, part of whose function is to control the electrolyte balance in the body. The zona glomerulosa forms desoxycorticosterone which acts to promote sodium retention and thus counteract the sodium depletion effect of the angiotonin. How-

[1] Page, I H "The Renin-Angiotonin Pressor System," Hypertension. University of Minn Press, Minneapolis, 1951, Page 48

[2] Zweifach, B W, and Shorr, E "Hepatorenal Vasotrophic Factors in Blood of Patients with Essential Hypertension, Factors Regulating Blood Pressure," Josiah Macy, Jr, Foundation, 1948, 137.

ever, desoxycorticosterone acetate acts not only to promote sodium retention but also causes the formation of nephrosclerosis which, in turn, increases renal ischemia on an organic basis which results in the elaboration of more renin and thus a vicious cycle is set up with an ever increasing hypertension as the result.

Davis, Tarkington and Anderson[1] have shown in experimental animals and in patients with essential hypertension, from whom biopsy specimens of the adrenal cortex have been taken before and after the administration of thiocyanates, that there is a definite depletion of the sudanophilic, or lipid, granules in the three layers of the adrenal cortex. The most striking change occurs in the zona glomerulosa. These changes in the lipid granules of the adrenal cortex return to normal when the blood level of the thiocyanates falls to zero and when the blood pressure rises.

That the liver plays a role in hypertension has been suggested by many investigators and to it has been assigned the role of the production of a vasodepressor substance which is normally in balance with a vasopressor substance originating in the kidney and adrenals. It has been demonstrated that the level of hypertensinogen in the blood plasma is maintained by production in the liver, and since the decrease in hypertensinogen is described only in some cases of experimentally acute liver injury, Davis and Tanturi[2] partially occluded the portal vein and produced low grade hepatic pathology. There was a delayed but permanent drop in blood pressure in the experimental animals with a coincident fatty infiltration of the liver but without damage to the liver cells. Under such conditions, it might be assumed that the liver would be unable to manufacture hypertensinogen or is no longer able to inactivate the vasodepressor substance.

Tumors of chromaffin tissue (pheochromocytoma), wherever it is found, but occurring most commonly in the adrenal gland, produce two types of hypertension. Paroxysmal hypertension, which is the most easily differentiated and diagnosed type, is characterized by an explosive, short-lived rise in blood pressure which returns to normal between attacks. However, after a number of such episodes, the blood pressure will drop following the attack but not as low as it was previously. Thus, a successively higher base line is established and other symptoms of excessive sweating, coldness of the extremities, pallor and numbness and other vasomotor symptoms occur. The second and often undiagnosed type, is the one characterized by a

[1] Davis, L., Tarkington, J., and Anderson, R. K.: "Physiologial Principles Underlying the Treatment of High Diastolic Hypertension by Thiocyanates and Sympathectomy," Ann. of Surg., 133, 867, 1951.

[2] Davis, L., Tanturi, C. A.: "The Liver as a Factor in Experimental Renal Hypertension," Arch. of Surg., 62, 325, 1951.

sustained hypertension and this is very similar to essential hypertension.

It is our custom to classify patients with essential hypertension into 5 clinical groups: (1) the *fluctuant* type which represents a large proportion of the early cases of hypertension observed usually in the teens or early twenties. It has often been described as adolescent hypertension and is not associated with renal disease. It is usually asymptomatic and is only discovered as the result of a routine physical examination. During these early years, it is impossible to tell whether the disease will remain quiescent, progress into one of the other clinical types, or in some cases, disappear entirely. As long as the etiology cannot be determined, associated renal diseases not discovered, and the patient remains asymptomatic, it is questionable whether or not any therapy should be instituted. However, in our experience, these patients uniformly respond well to the administration of the thiocyanates.

The *plethoric* group is the second type. The plethoric hypertensive is usually the typical "businessman" with hypertension. The patient is energetic, active, overweight and may have distressing symptoms, particularly headaches, vertigo and angioid distress associated with cardiovascular disturbances. The hematocrit is usually found to be over 50 per cent; the red blood count over 5 million and the sedimentation rate 1 to 5 millimeters or less in one hour. We are in the habit of describing the blood as viscid, or "sticky," and in such cases, cerebral or coronary thrombosis are common. Provided that these patients do not have associated irreversible renal disease, continued control of a blood cyanate concentration will result in a rather prompt relief of the hypertensive symptoms and a gradual diminution of first the systolic, and later, the diastolic pressure. During the first few months of treatment with the thiocyanates, the hematocrit will drop to 45 or less and the sedimentation rate will increase to 10–15 mm. in an hour. This represents a definite change in the hemogram resulting from the thiocyanate effect on the red blood count and, therefore, on blood viscosity.

Menopausal hypertension is one of the most frequent types and constitutes the third group It corresponds in many respects to the plethoric type of hypertension in the male. It is accompanied by numerous and severe symptoms of hypertension which are superimposed upon those which are associated with the menopause However, a high hematocrit reading [illegible] ow sedimentation rate are

to thyocyanate therapy. It should not be inferred that the menopause is, in itself, a cause of essential hypertension. The menopause may exaggerate the symptoms of a pre-existing, or coincidental, hypertension in patients with only a moderate elevation of the blood pressure. It has long been observed that if the patient can be brought safely through the period of menopause, be it five or fifteen years, the hypertension may subsequently burn itself out.

The fourth group includes the high diastolic type of hypertension as it is seen in the male, most often between the ages of thirty-five and fifty. It represents the most severe type of essential hypertension. It is indicative of either a pre-malignant phase of hypertension, or a rapidly progressing type of essential hypertension in which the occurrence of heart failure, or a cerebral vascular accident, within a five-year period is usual. Unlike the female in this same age group with a severe high diastolic pressure, the male does not tolerate an increased blood pressure for a very long period of time. These patients may not respond well to the thiocyanates except for irelatively short periods of time and if they do show some blood pressure lability and some response to the thiocyanates, though it is temporary, we have subjected them to sympathectomy before the onset of renal and cardiac failure, or malignant hypertension.

Group 5 includes arteriosclerotic hypertension, usually seen in its early phases between the ages of fifty and sixty, characterized by relatively high systolic and relatively low diastolic blood pressure readings. Its course well represents the prolonged and benign nature of some cases of hypertension, although it is often accompanied by cerebral symptoms of headaches, vertigo, tinnitus, and even symptoms of left ventricular failure. The disease itself is not severe and is compatible with a rather long, useful life. Some patients respond very well with small doses of thiocyanates but they should be observed very carefully in order to avoid a rapid drop in blood pressure.

The final group is that of malignant hypertension, characterized by severe hypertensive symptoms, including convulsions and coma. It may occur in any age group and its clinical course if rapidly progressive to a fatality. It mainfests itself by a severe, progressive, generalized necrotizing, arteriole disease and renal failure. The disease shows no response to the thiocyanates or any other medicinal type of therapy. A patient with any of the previous types of hypertension may suddenly enter the malignant phase of essential hypertension and this occurrence is unpredictable and most likely to happen in the high diastolic group.

Surgical Treatment.—Three types of operations have been used in the surgical treatment of essential hypertension: rhizotomy, sympathectomy and adrenalectomy. Interpretation of results has

been slow, and justly so, in view of the unpredictable and protean nature of the disease as well as the time required to determine whether its course may or may not have been significantly benefited by operation.

Because the results on the blood pressure and the ultimate outcome following removal of the second, third, and fourth lumbar sympathetic ganglia with the intervening chain were not significant in operations performed for the treatment of malignant hypertension in 1925, and in order to secure complete sympathectomy below the diaphragm, Adson,[1] in 1930, performed a bilateral section of the anterior and posterior roots from the 6th thoracic to the second lumbar segment of the spinal cord inclusive. Later Brown, Craig, and Adson[2] reported a series of 8 cases treated by bilateral anterior rhizotomy from the 6th thoracic to the 2nd lumbar roots with an effective permanent depression of the blood pressure. In fact, the upright posture caused such a terrific sudden drop in blood pressure that abdominal binders were necessary in some cases. Page and Heuer[3] reported on a series of 20 patients treated by section of the anterior roots whose conditions varied from benign essential to almost highly malignant hypertension.

As early as 1929, partial removal of the adrenal gland was performed by various foreign surgeons and by DeCourcy in the United States. In an early report DeCourcy compared subtotal thyroidectomy for hyperthyroidism to bilateral subtotal removal of the adrenal gland for hypertension.

At this time the trend of surgical procedures swung toward adrenal denervation by celiectomy as practiced by Crile, or by splanchnic nerve resection as practiced by Peet and others. Whereas Adson resected the lumbar sympathetic ganglionic chain, Peet resected a portion of the 3 splanchnic nerves and the chain and ganglia from the 10th thoracic ganglia downward to the diaphragm. Because of the impression that both the supradiaphragmatic and the subdiaphragmatic methods of resection of the splanchnic nerves and the sympathetic ganglions had their advantages and that a more extensive denervation would be followed by a greater response

[1] Roundtree, L. G., and Adson, A. W.: "Bilateral Lumbar Sympathetic Neurectomy in Treatment of Malignant Hypertension," Jour. Am. Med. Assn., 85, 959, 1925.

Adson, A. W., Brown, G. E.: "Malignant Hypertension; Report of Case Treated by Bilateral Section of Anterior Spinal Nerve Roots from 6 Thoracic to 2 Lumbar Inclusive." Jour. Am. Med. Assn., 102, 1115, 1934.

[2] Brown, G. E., Craig, W. M., and Adson, A. W.: "Treatment of Severe Essential Hypertension. Effects of Surgical Procedures Applied to Sympathetic Nervous System." Minnesota Med., 18, 134, 1935.

[3] Page, I. H. and Heuer, G. J. "Treatment of Essential and Malignant Hypertension by Section of Anterior Nerve Roots." Arch. Int. Med., 59, 245, 1937.

the two were combined into the transdiaphragmatic type of surgical procedure by Smithwick. In fact, removal of the sympathetic chain and ganglia from the 1st thoracic to the 4th and 5th lumbar ganglia has been advocated on the basis that *the lowering of the* blood pressure and relief of symptoms following sympathectomy *has been directly proportional to the extent of the sympathectomy* and inversely proportional to the severity of the disease.

The multiplicity of operations which have been tried and the controversy which has been aroused with regard to the application and to the results of surgery is evidence that no one procedure has proved infallible. In the beginning, the operations were based upon the assumed physiological objective that denervation of the vascular tree of the abdominal viscera and the lower extremities by a sympathectomy would significantly change the course of the hypertension. On the other hand, those advocates of removal of a portion of the adrenal gland believe that sympathectomy denervates the adrenal medulla, although in the beginning the explanation of the results thus obtained was not possible.

It is our belief that operation should be reserved for those patients with essential hypertension who have been given an adequate and thorough therapeutic trial with all types of medicines including the thiocyanates. If the patient's symptoms including his diastolic pressure cannot be improved, then surgical treatment should be considered before irreversible changes in the vessels of the heart, kidneys, brain and other organs occur A third indication for the choice of patients for surgery, we believe, is that they should have a labile blood pressure as evidenced by a group of pressor and deep pressor tests. The patient should have no evidence of chronic glomerulonephritis or pyelonephritis, no significant peripheral vascular sclerosis, and the clinical course should be shown to be progressive, but not entering the malignant stage of hypertension. We have used sodium amytal, sodium pentothal, caudal anesthesia, induced hyperpnea, tetraethylammonium chloride and vertavis as vaso-relaxor tests, and the cold pressor test and the administration of carbon dioxide as vasopressor indicators. However, no one of these tests, or any combination of them, have ever given us any information of the character, or extent of the response we might expect after sympathectomy.

In 1947, Peet[1] reported on bilateral supradiaphragmatic splanchnicectomy in over 1500 patients, most of them followed one to twelve years, and stated that five to twelve years after operation 81.3 per cent of those patients still alive had a significant drop in both systolic

[1] Peet, M M "Results of Bilateral Supradiaphragmatic Splanchnicectomy for Arterial Hypertension," New England J. Med., 236, 270, 1947.

and diastolic pressures. Life expectancy had improved in 19 per cent of those with preoperative malignant hypertension who were still alive five to thirteen years after operation, compared with less than 1 per cent in a comparable series of patients treated medically. Poppen and Lemmon[1] reported on 100 consecutive cases of thoraco-lumbar sympathectomy performed for hypertension that had existed for one and one-half to four years. They were unable to find any valid tests for selecting patients for sympathectomy and stated that symptomatic relief was found to occur even if the blood pressure was not reduced.

Hoobler and his colleagues[2] reported on 294 patients with essential hypertension, studied before and 1 year after supradiaphragmatic splanchnicectomy and lower thoracic sympathectomy. They concluded that 29 per cent of these surgically treated patients had reduction of blood pressure outside the range of spontaneous variation over this period. They also noted that if the blood pressure remained at a low level for as long as one year, the chances of its remaining permanently decreased were greater.

Thorpe, Welch and Poindexter[3] followed 500 patients on whom thoraco-lumbar sympathectomy was done. They reported excellent results in 11.8 per cent of the patients but they noted no significant increase in survival over those treated medically, except in the severely hypertensive groups. Whitelaw and Smithwick[4] reported upon a study of 500 patients operated upon and followed for a year or more, in which 36 per cent had a drop in the diastolic pressure of 20 millimeters of mercury or more, and a total of 67 per cent showed some postoperative decrease in pressure. This was compared with a control group treated medically who showed a lower diastolic pressure in only 25 per cent of the patients.

While there have been many reports as to the results of surgery in regard to blood pressure and symptoms, there has been but little information available concerning the effect of surgical treatment upon life expectancy Smithwick[5] has reported the mortality and survival rates after thoraco-lumbar sympathectomy in the first 892 consecutive cases followed for a period of from four to twelve years. It

[1] Poppen, J L, and Lemmon, C. "The Surgical Treatment of Essential Hypertension." Jour Am Med Assn, 134, 1, 1947

[2] Hoobler, S W, Manning, J L, Paine, W. G., McClellan, S. G., Helcher, P O, Renfert, H Peet, M. M. and Kahn, E A. "The Effects of Splanchnicectomy on the Blood Pressure in Hypertension." Circulation, 4, 173, 1951.

[3] Thorpe, J J, Welch, W J, and Poindexter, C A. "Bilateral Thoraco-Lumbar Sympathectomy for Hypertension." Am J. Med, 9, 500, 1950

[4] Whitelaw G P, and Smithwick, R H "Effect of Extensive Sympathectomy upon Blood Pressure Responses and Levels." Angiology, 2, 157, 1951.

[5] Smithwick, R H "Hypertensive Cardiovascular Disease." Jour. Am. Med. Assn, 147, 1611 1951

would appear that there is a real significant increase in life expectancy made possible by surgical intervention in all types of essential hypertension graded as to their severity. It would not appear from the reports in the literature that total sympathectomy, as first advocated by Grimson in 1941, materially improves the results of a less extensive operation. Our own experience would corroborate this fact because we have found no difference in the immediate or late result of the operation regardless of the extent of the sympathectomy upward or downward providing the area of sympathetic supply to the adrenal glands is included in the resection.

Recently, removal of the adrenal glands either completely or partially has been brought to the attention of those attempting to treat this disease. Zintel and his colleagues[1] have reported a series of 26 patients of whom 13 had partial adrenalectomy and 13 had a partial adrenalectomy plus a sympathectomy. Eleven patients were followed for a period of from four to twelve months following operation. Three patients died following the operation, 2 shortly thereafter, and 1 in six weeks. One patient has had a normal blood pressure for 1 year following operation. All of the patients have required very careful replacement therapy and it has been noted that there is a small margin between the amount of the replacement needed to control Addison's disease and the amount which results in the return of the hypertension. These same investigators have also stated that unilateral adrenalectomy plus thoraco-lumbar sympathectomy has not produced results any better than one might expect from a sympathectomy alone.

We have found that sympathectomy, regardless of the extent of the operation, providing the adrenal glands are denervated, materially affects the result of the treatment with the thiocyanates. That is to say, that patients who have been resistant to the administration of the thiocyanates before sympathectomy have shown very favorable reaction to this drug following sympathectomy. It has been our purpose clinically and experimentally to attempt to find a physiological explanation for this clinical fact which has been corroborated by others. Hypothetically, it may be suggested that the thiocyanates deplete the zona glomerulosa of the adrenal cortex which is assumed to be the place of origin of the ketosteroids. Further that sympathectomy denervates the adrenal medulla which in turn through the action of the pituitary gland depletes the adrenal cortex. Thus, the thiocyanates and sympathectomy act to reinforce each other. In mild cases of hypertension either the thiocyanates or any

[1] Zintel, H. A., Wolferth, C. C., Jeffers, W. A., Hafkenschiel, J. H. and Lukens, F. D. W.: "Subtotal Adrenalectomy in the Treatment of Patients with Severe Essential Hypertension" Ann. Surg., 134, 351, 1951

other type of medicinal therapy may result in a very favorable clinical course just as a sympathectomy will produce the same result. However, in more severe cases either the operation alone or the thiocyanates alone are not sufficient to balance the overproduction of the pressor substances.

Essential hypertension is one of the leading causes of death and is four times as deadly as cancer. In many patients, it causes as much discomfort and suffering as does cancer. It may well occur that the present surgical treatment of essential hypertension may be replaced by the discovery of medical methods which will restore the normal balance between pressor and depressor substances in the blood However, in the meantime, there is enough evidence that this surgical procedure either alone or combined with medicinal treatment provides a method for lessening the effects of this disease.

CHAPTER XII

THE SURGICAL TREATMENT OF CONVULSIVE SEIZURES

"Epilepsy is an experiment made upon the brain by disease."—
HUGHLINGS JACKSON

THOUGH the diagnosis of epilepsy in most of its multiple and varied manifestations is easy, its underlying nervous system physiology is obscure and difficult to comprehend. That epilepsy is not a disease is agreed upon. The clinical entity is the result of a sudden, violent discharge of neural elements produced by a noxious stimulus.

There is a form of epilepsy, by some termed "idiopathic" or "true" epilepsy, which has as its only symptom convulsive seizures. No anatomical substratum has ever been discovered for this type of epilepsy. Then there are diseases which have convulsive seizures as a part, and often only a small part, of their symptomatology; for example, intracranial tumors, abscesses, arachnoiditis, cerebral arteriosclerosis, intoxications, cerebral dysplasias, vascular anomalies, cerebral degenerative states, and traumatic alterations of the brain. Such epileptiform seizures must be differentiated from convulsions, the etiology of which cannot be determined. It is with the pathological conditions accompanied by epileptiform or convulsive siezures that surgical therapy is concerned.

An epileptiform seizure may include symptoms the result of activity of the cortical motor area; the so-called sensory area; or the cerebral visual, auditory, olfactory, gustatory, and autonomic centers. In accepting such a conception, there is no difference between a localized motor spasm, paresthesia of a limb, hallucinations of sound, smell or sight and certain visceral and autonomic phenomena. All are epileptiform seizures, which may usher in a complete convulsive attack, and under such circumstances may be regarded as an aura.

The continued search for the physiological facts of epilepsy has led to acceptance of the basic unity of the convulsive state as well as to the astonishing variety of stimuli which may produce such episodes. Though the symptoms be protean in character and the nature of the stimulus varied, it is probable that there is a common, connecting neural mechanism.

Since the discovery by Fritsch and Hitzig of the electrical irritability of the brain, numerous theories concerning the site of convulsive discharge have been proposed. It is unfortunate for the clarity of our knowledge that this site has been considered inseparably from the pathogenesis of the seizures. It is further unfortunate that much of the work has been concerned with the establishment of a so-called convulsive center.

Three chief theories have been proposed concerning the location of a convulsive site. First, that which deals with the cortex alone; second, that which includes the cortex and subcortex; and third, the medullary theory. The experiments performed to prove any of these theories have been of two types; first, those in which convulsive seizures have been produced by irritation of various parts of the central nervous system and second, those in which experimentally produced convulsions have been modified, stopped or prevented by ablation of certain parts of the nervous system.

The proponents of the medullary theory have assumed that if convulsions may be produced by irritation of the pons and medulla, and may occur as the result of intoxication with absinthe in decerebrated animals, the convulsive site either exists in the pons and medulla, or the pons and medulla possess epileptogenous properties. Opposed to this theory are the observations that when a convulsion occurs as a result of irritation of the pons, the clonic element disappears after removal of the cerebral cortex; and that removal of one cerebral hemisphere causes convulsions to disappear from one-half of the body.

Those who support the mixed theory are dependent upon observations of certain motor manifestations produced by stimulation of various parts of the central nervous system and many of these experiments have nothing to do with convulsions. They are concerned with the production of certain forced movements, ataxia and dystonia.

The evidence which has supported the theory of cortical origin has been supplied chiefly by the observation of convulsions produced by electrical stimulation of the cortex. It has been found that convulsions may be produced which resemble epilepsy It has also been shown that if electrical stimulation of the motor cortex is long continued, the resulting isolated contractions of muscle groups become successive and spread to muscle groups innervated by adjacent cortical areas; then spread to the other side with the resulting convulsion outlasting the electrical stimulation If an irritated zone be extirpated quickly, the convulsion is inhibited After deep decortication, such convulsions cannot be produced and when certain motor areas have been removed, the movements represented by such an area are absent in an ensuing convulsion. Not only can such convulsions be produced by irritation of the motor cortex, but by stimulation of other cortical areas such as the occipital lobe

From our own studies,[1] and from the accumulated reports of experimental work, it may be stated that whatever element of a

[1] Pollock, L. J., and Davis, L.: Experimental Convulsions, Arch Neurol and Psychiat 20, 756, 1928

seizure may be lacking in a convulsion produced in an animal with a mutilated brain, convulsions (tonic, clonic, or both) may be produced from the cortex and subcortical region and, in the latter case, in the absence of the motor cortex. The symptoms of a convulsion vary with the state of preservation of the brain. If it be intact, tonic-clonic convulsions associated with sialorrhea, pupillary changes, bladder contraction, circulatory and respiratory changes, and at times an aura may be present. The aura is dependent upon the area of brain irritated; and in Jacksonian attacks, as in artificially produced cortical seizures, motor movements constitute the aura. In other cases, a vagal aura exists, respiratory or circulatory changes precede the convulsion, and the portion of the brain irritated must be that of a lower level. It may be pointed out that not only is the tonic element of an epileptiform seizure absent in a cortical fit, but loss of consciousness does not occur until a generalized convulsion ensues, and involuntary urination does not occur before the loss of consciousness. A major epileptic convulsion consists not only of tonic and clonic movements but also of a series of events including the aura, vascular, respiratory, secretory changes, and contraction of the involuntary muscles of the bladder.

The earliest proved adventure of man into the field of major surgery was trepanation for fits, and so it may be said that man's experience with the surgical treatment of the convulsive state began at least a quarter of a million years ago. Though the progress of surgical therapy waxed and waned, the problem of epilepsy has remained persistently for solution. The contribution of Hippocrates to the treatment of injuries of the head gave rise to a literature dealing with the surgical aspects of traumatic convulsions which grew to an enormous proportion by the time of the sixteenth century. To Guy de Chauliac epilepsy was simply a convulsion of the entire body arising from excess of humidity and "it suffices the surgeon to know what has been said of them, their symptoms and cure if there is any." The character of these surgical procedures may be judged fairly well when it is realized that even during the seventeenth century Thomas Willis struggled valiantly and alone to wean his more philosophical colleagues from an unshaken belief in the rôle of Satan in convulsive states by constantly centering their attention upon the central nervous system.

Certain general principles influence a clinical study of patients with convulsive seizures, for example, What role does the so-called "epileptic constitution" or "make-up" of the patient play in an evaluation of his seizures? Why do convulsions occur in one patient with an intracranial tumor and not in another patient with a tumor similar in type and location? Similarly, does not the "potentially

an epileptic" factor play an important role in the development of convulsions in the approximately 5 patients out of 100, all of whom have received severe wounds of the brain?

Obviously it is not entirely satisfactory to classify the location of the pathological changes on the basis of what is observed in operation. Adjacent portions of the brain may be involved in the absence of gross changes in appearance. With proper emphasis upon this reservation, the operative records and sketches, with the exception of cases in which an autopsy is performed, may constitute the basis for localization.

In a study of our own material, all patients with symptoms which to us represented epileptiform manifestations, or a convulsive state, were included. Periods of sudden unconsciousness, not syncope; periods of twitching or drawing of the hand or foot; peculiar odors with periods of apprehension are as significant to us as major seizures; *in fact, many of these minor spells are of more localizing and diagnostic value.* However, many reports in the literature upon the occurrence of convulsions in groups of patients with intracranial neoplasms exclude this important group.

It is exceedingly difficult to obtain an accurate description of a seizure. To describe all of its manifestations, if one has the opportunity to observe an attack, is often impossible. Patients are told what happened after they lost consciousness and many important details of the symptoms are lost. Patients, or relatives, may regard or neglect minor manifestations as insignificant in the presence of more dramatic symptoms.

It is difficult, if not impossible, to compare one case directly with another. It is of more value to compare the condition of the patient before and after operation, with and without medical treatment. Is the patient as well off after operation as he was before operation? Can it be said in a given case that the surgical procedure made it possible to control the convulsions more successfully by adequate medicinal therapy?

It is somewhat of a surprise to realize the large number of patients with intracranial tumors who have convulsions and particularly the large number in whom a convulsion was the initial symptom which first called the attention of the patient or his family to his difficulty. It was not long ago that the general impression existed, and it was taught, that the meningiomas were the intracranial tumors most often associated with convulsions. In our own experience, 62 per cent of the patients with glioblastomas, 72 per cent of the patients with astrocytomas, 39 per cent of the patients with meningiomas, and all but one patient of the angioma group presented epileptiform seizures. Four patients of 54 who had pituitary tumors had con-

vulsions, but it should be noted that in each of these patients the tumor had grown outside of the boundaries of the sella turcica. About 40 per cent of our patients with metastatic intracranial tumors presented attacks of one type or another.

Reports from other clinics vary from an occurrence of convulsive seizures in tumors of the brain from 25 per cent to 51 per cent in intracerebral and 62 per cent in extracerebral neoplasms as reported by Penfield.

In any series of intracranial tumors, the majority of patients are adults. It is important, therefore, to point out and to re-emphasize the fact that *convulsions occurring for the first time in adult life should be immediately suspected of being due to an intracranial tumor.* It remains, therefore, a duty of the physician to bear this in mind and to use every possible diagnostic aid at his command to prove that this patient does not have a space-occupying lesion.

Convulsive seizures may be present long before there is evidence of increased intracranial pressure, a symptom which many physicians believe must be present before the diagnosis of a space-occupying lesion can be made. It cannot be repeated too often that to prove that the patient does not have a space-occupying lesion is the duty of the physician and not of the individual.

Our material was studied to determine whether or not any relation existed between the occurrence of epileptiform seizures and the location of the space-occupying lesion. Quite in keeping with what our general ideas have been from our clinical experiences and experimental investigations, we found that seizures occurred in patients with tumors which were situated in the cortex, subcortex, pons, midbrain, third ventricle, and posterior fossa. The only tumors which occur in the posterior fossa and which were never accompanied by convulsive seizures in the patients in our series were the acoustic neurinomas.

An attempt was also made to relate certain epileptic manifestations with definite functional areas in the brain. It should be immediately recognized that this is just as difficult as it is to draw conclusions concerning localization from tumor or abscess pathological material. Both at operation and autopsy it is hard, particularly in the gliomas, to determine the limits of the tumor growth. Although a meningioma does not involve the brain directly, the extent of damage produced by pressure is equally as difficult to determine. So, we have been able only to make certain rather general deductions in relation to gross divisions of the brain. After analyzing all of the cases for objective and subjective phenomena presented in the described seizures, a very few facts stand out. For example, as compared to the frontal, temporal and the occipital lobes, localized

muscular twitchings, commonly described as a Jacksonian attack, occurred far more often in those tumors situated in the parietal lobe. On the contrary, when such objective symptoms as pallor, drooling, cyanosis, flushing, or lacrimation occur, all of which might be grouped as vasomotor symptoms, they were found to be present in tumors in the frontal or temporal lobes and not in tumors of the parietal or occipital lobes Loss of consciousness occurred just as often in the tumors of one lobe as of another. We cannot subscribe completely, therefore, to the view that arrest of consciousness occurs when the frontal lobe alone is discharging.

Likewise, it is obvious from our material that the subjective epileptic manifestation of a bad odor or taste occurred exclusively in tumors of the temporal lobe and, more particularly, in tumors which involved the medial aspect of that lobe, the uncinate gyrus. The subjective symptom crudely described by the patient as "tingling, numbness, or electric shock" occurred more often in parietal lobe tumors situated within or posterior to the postcentral convolution; but in two instances of frontal lobe and in the same number of temporal lobe tumors, the patient described a similar sensation preceding his attack. Here, of course, lies the danger of stating that this symptom occurs in temporal or frontal lobe tumors because only by careful pathological examination of the entire brain could one be justified in stating that the postcentral convolution of the parietal lobe was not involved by the tumor. Such a circumstance could also be explained, of course, by an involvement of one of the large cerebral arteries so that an epileptiform seizure might begin in any portion of the brain at a distance from the neoplasm but in the area of distribution of the artery We have seen temporary complete blindness during the discharge of the occipital lobe produced by a tumor, and this has been demonstrated by others.

Penfield[1] has reported the occurrence of "autonomic epilepsy" which occurred in a patient with a tumor of the third ventricle It *would seem from this case that a convulsive discharge may* occur from centers as high as the anterior portions of the thalamus. The reports of other patients with sensory epileptiform seizures in which the tingling spread gradually through one side of the body and became so severe as to be painful also suggests that the gray matter of the thalamus may become involved even if the original discharge does not take place there.

It has been our experience that immediately preceding a convulsive seizure deviation of the head and eyes occurs toward the side of the lesion, that is, toward the site of irritation This is not in

[1] Penfield, W Diencephalic Autonomic Epilepsy, Arch Neurol. and Psychiat., 22, 358, 1929

accord with statements of some individuals who feel that the most frequent lateralization sign is deviation of the head and eyes to the side opposite the hemisphere involved. It is important to attempt to localize epileptic manifestations within the brain so that convulsive seizures can be accurately interpreted, as an aid both to diagnosis and therapeusis, but we doubt very much whether or not definite epileptiform manifestations can be ascribed solely and specifically to certain lobes or gyri of the brain.[1] However, we are able to confirm the clinical fact which has been known for some time that the epileptic manifestations of a tumor in the posterior fossa are very definite. The outstanding characteristics of these convulsive states is the occurrence of a tonic convulsion involving all four extremities, opisthotonos, with marked respiratory and vasomotor symptoms. Psychomotor seizures, characterized by confusional episodes in which the patient may perform apparently purposeful but coarse and poorly co-ordinated movements, have been stated to be associated with a focus in one or both anterior temporal lobes.[2] The evidence for this localization has been based almost entirely on electroencephalographic tracings which reveal seizure discharges in a high percentage of the patients when the tracing is made during natural or induced sleep.

When one begins to determine the effect of removal of an intracranial tumor upon the occurrence of convulsive attacks, several factors arise which interfere with the collection of data of accurate scientific value. It is not always possible to determine the frequency or the nature of seizures in a given patient before operation under the conditions of accurately controlled therapy, or without therapy. Likewise, it is not always possible after operation to allow a patient to go for a period untreated with medicine in order to make a comparison with his condition under treatment. It is very seldom that the patient with epileptiform seizures is observed over a sufficiently long period before operation by the surgeon who removes his intracranial tumor. This introduces the factor of the judgment of several clinicians as to severity, frequency, and nature of the attacks before and after operation.

We attempted to study all of our intracranial tumor patients in an effort to determine whether the attacks were more frequent after operation, whether they remained the same, whether they were less

[1] Investigations with the electro-encephalograph have been made upon patients with epileptiform seizures. It has been stated by Lennox (Arch. Neurol and Psychiat., 44, 1156, 1940) that the constant feature of the tracings of a patient with various types of seizures may be the inconstancy of the rate of voltage of the waves of cortical activity.

[2] Bailey, P. and Gibbs, F. A: "Surgical Treatment of Psychomotor Epilepsy," Jour Am Med. Assn., 145, 365, 1951.

in frequency, or whether they did not recur. In 1936, of the patients with glioblastomas, and it must be remembered that the life expectancy of patients with this type of tumor after operation is on the average of six months to eighteen months, 10 per cent of those having attacks prior to operation had none after operation up until the time of death, and 32 per cent had fewer and less severe attacks following operation. Of the astrocytoma group, 31 per cent were judged to be unimproved as far as their convulsions were concerned. Fifty per cent had fewer and less severe attacks, and 6 per cent had no attacks following operation over an interval of three and six years, respectively. These figures represent about the condition which exists in the other tumors in the glioma group.

Of the meningioma group, and in these patients the tumor was removed completely, 3 patients were definitely worse following operation and finally had to be placed on anticonvulsant therapy. Eight patients had fewer attacks which were less severe but were not entirely free in spite of the fact that their tumors had been completely removed. The remaining 5 of the 16 patients in the group who had convulsions before operation had no further convulsions following removal of their tumor and did not receive therapy. It becomes apparent that although these tumors were removed completely certain changes must have been produced within the brain which continued to act as irritative foci which released a convulsive discharge. These changes may be and probably are microscopic in nature and further surgery under such circumstances is illogical and contra-indicated. Particularly is this true when the attacks can be so well controlled by judicious medicinal therapy.

In general, one may say without a doubt that the removal of an intracranial tumor is followed in the majority of patients by definite improvement in the frequency and severity of the convulsive seizures. It cannot be guaranteed, however, that the attacks will completely disappear. It is now our custom to begin anti-convulsant therapy preoperatively upon those patients who give a history of convulsive attacks and to continue that therapy after operation.

As our records show, successful drainage of a brain abscess may be followed in later years by the development of epileptic seizures and the pathological changes which occur in the brain during the obliteration of the abscess cavity provide a focus from which the convulsive discharge may occur.

A second group of patients with convulsive seizures are those in whom injury to the brain has played an important rôle. Here again, before the exact rôle of trauma can be evaluated in each case a study should be made to determine what part the constitutional make-up of the individual has played in the development of the

epilepsy. This is particularly emphasized by the statistics which have been gathered by a number of men relating to the occurrence of epileptiform attacks following trauma to the brain. In a group of 18,000 cases of gunshot wounds of the head, Turner found that less than 5 per cent of the patients developed seizures. Ascroft[1] found that of 317 cases of craniocerebral trauma, 107, or 34 per cent, later developed epileptiform seizures. He found that seizures developed twice as frequently in those cases in which the dura mater was penetrated than in those in which the dura mater remained intact. He also concluded that removal of metallic foreign bodies apparently did not diminish the occurrence of seizures, but on the contrary, wound sepsis increased the occurrence of seizures.

It has been well established that when the brain is injured and the damaged cerebral tissue is not removed, a scar of fibrous astrocytes, connective tissue and newly-formed blood-vessels develops. This scar soon becomes densely adherent to the overlying meninges. The rich plexus of blood-vessels anastomose with the cerebral vessels on one side and with the vessels of the meninges and scalp on the other. Like all cicatricial tissue, this scar contracts and a pull upon the frame work of glial and vascular tissue develops. This cicatricial pull is not confined to the local area of scar tissue; the homolateral hemisphere and even the entire brain is affected.

Foerster[2] has described the mechanism of the pull of such a cicatrix. He noted a displacement of both lateral and third ventricles toward the site of the scar tissue with enlargement of the lateral ventricle upon the side of the lesion. There is a certain amount of cerebral atrophy which accompanies the formation of scar tissue, but it probably plays no important rôle in the ventricular displacement.

Penfield's[3] examination of the scar tissue removed invariably showed fibrous tissue near the surface of the cortex and meningeal adhesions. The fibers of astrocytes were arranged parallel to the direction of traction and extended upward toward the scar. Deep within the brain the astrocytes and blood-vessels formed the only framework capable of resisting the constant pull of the contracting cicatrix. In cases of long-standing, tubes and sheets of neuroglia surrounded by connective tissue sheaths may result from the progressive cicatrization. We have seen gelatinous, yellowish-gray fibrous tissue as the center of an area of atrophy and cyst formation in old

[1] Ascroft, P. B: Traumatic Epilepsy After Gunshot Wounds of the Head, Brit. Med. Jour., 1, 739, 1941.

[2] Foerster, O.: Encephalographische Erfahrungen, Ztschr. f. d. ges. Neurol. u. Psychiat., 94, 512, 1924.

[3] Foerster, O., and Penfield, W.: The Structural Basis of Traumatic Epilepsy and Results of Radical Operations—Epilepsy and the Convulsive State, Baltimore Williams & Wilkins Company, p 587, 1931.

cases; a fact which Foerster and Penfield record, emphasizing the focus of concentric traction.

The pathogenic relation between the formation of the scar, the development of a vascular plexus, the steady pull of the contracting scar and epileptic attacks is immediately suggested. Foerster and Penfield have expressed the problem very clearly:

"If the stimulus for an epileptic seizure arises in the nervous elements one might expect the attack to begin shortly after the infliction of the wound when these elements are plentiful and undergoing progressive destruction If, on the other hand, the vascular elements are responsible for the initiation of the process, epilepsy might well be expected to make its appearance later, when the vascular plexus reaches its fullest development and nerve-fibers have largely vanished.

"It has been shown that at operation the focal epileptic attacks may often be produced in two ways; either by electrical stimulation of the brain in the neighborhood of the wound or by gently pulling upon the adherent dura This latter fact may be of considerable significance for, if the increase of a pre-existing strain produces an attack, it may well be that the pre-existing strain itself is an important factor in the etiology of spontaneous convulsions.

"As pointed out above, the blood-vessels form in one sense the roof of the

The radical excision of one of these extensive, contracting scars for the relief of convulsions, assuming that the mechanism just described constitutes the pathogenesis of the attacks, can only be a rational procedure if the damage produced by excision is not accompanied by the same train of histological events that followed the original lesion. Penfield's histological studies[1] made upon the excision of cerebral tissue would point to an entirely different result. He has stated that when the brain tissue is excised, there is no organization of connective tissue. Instead, a fluid-filled space with a small amount of gliosis about it appears. There is no plexus of newly-formed vessels, and there is no evidence of a cicatricial pull upon the surrounding brain tissue. Brain injury and brain excision are followed by entirely different structural changes. (Fig. 172)

If this were the entire story underlying the surgical relief of epileptiform seizures, the problem would be relatively simple. Only the careful study of patients, as human experimental problems, will answer the question of the wisdom of substituting a non-contracting fluid-filled space for a contracting cicatrix. One must assume, in such a working hypothesis, that removal of an apparently gross area of irritation will cause the entire neural mechanism responsible for a convulsion to disappear. This question has not been answered

[1] Penfield, W The Mechanism of Cicatricial Contraction in the Brain. Brain, 50, 499 1927

as yet by clinical cases carefully studied over a sufficiently long period. However, a valuable suggestion for the prophylactic treatment of severe brain and skull injuries has been made. The careful painstaking débridement of compounded cranio-cerebral injuries might reasonably be expected to decrease the incidence of post-traumatic convulsive attacks. (Fig. 173.)

There has been a widespread use of tantalum, or acrylic resin, plates to repair the defects resulting from severe cranio-cerebral injuries suffered in World War II. Many of the skull defects have

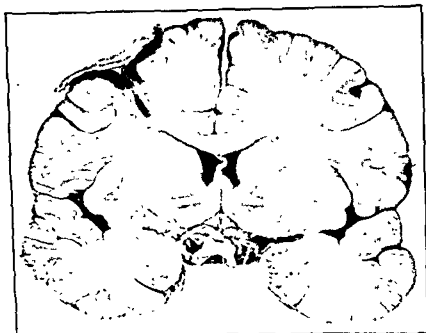


FIG. 172.—A coronal section of the brain of a patient with traumatic epilepsy. Note the thickened dura mater and the cerebral scar which pulls the lateral ventricle toward it.

been repaired without a thorough neurological exploration of the dura mater or underlying cerebral cortex. In fact, more often than not, air studies of the ventricular and subarachnoid systems have not been made. In some instances, however, pre-operative air studies have been made routinely upon any patient who has been considered a candidate for repair of his skull defect. Whether or not the repair of skull defects comparatively soon after injury will prevent the later occurrence of epileptiform seizures is not known. It would seem that if the cortical scar pulls the brain toward the surface, the dura mater should be opened and the brain released before the skull defect is repaired. It will remain for a careful, painstaking study of these patients, carried out over a number of

years, to answer this question, but it should be one of the contributions of war to the progress of surgery. It is also true that patients who have received cranio-cerebral injuries with a resulting skull defect unanimously desire its repair because as they express it, "they feel safer," and in dealing with military personnel this factor must be considered.



Fig. 173.—Photomicrograph of the thickened meninges and the cortical scar shown in Figure 172.

In 1936 we summarized our experiences with post-traumatic seizures which were purposely limited to those individuals who had organic evidences of the severity of their original injury; those who had organic symptoms following their convulsions and those in whom encephalographic studies demonstrated a mechanical defect which surgical intervention might be expected to remedy. The material included 13 patients whose attacks were attributed to cranio-cerebral compounded wounds due to blows on the head by bricks, pipes,

hammers; bullet wounds or automobile injuries; 4 patients with porencephalic cavities some of which were associated with a congenital skull defect; 1 patient with a definite history of birth injury; and 5 patients with convulsions which followed encephalitis, all of whom had focal attacks with organic symptoms of the lesion.

In contrast to the 13 patients with severe compounded cranio-



FIG 174.—Ventriculogram which shows the communication of the right lateral ventricle with a large porencephalic cavity in a child with Jacksonian convulsions.

cerebral wounds with convulsions, we observed and treated 32 other patients all of whom had gross cerebral damage and none of whom had then developed convulsive seizures. However, this time limit apparently never runs out. Six of the 13 patients had an interval of over six years between the injury and their first attack. The longest interval was twenty-one years and the shortest six months.

At operation several varieties of pathological changes were observed in this group of cases. In all of those in which there had been gross damage to the scalp, skull and brain, dense adhesions were found

between the dura mater, brain and skull. In several instances the skull defect had caused the underlying brain to become firmly attached to the scalp. As the scalp flap was elevated, the scar tissue pull would bring the brain along with it. We found all grades of arachnoidal thickening, often so opaque that the cerebrospinal fluid accumulated in lakes along the sulci between the convolutions. Often the fluid so trapped was yellow in color, further evidence of the lack of circulation in the subarachnoid space

Increased vascularity of the dura mater and cicatrized brain tissue was a constant finding. In one patient who had received a stellate-shaped depressed fracture of the temporal bone twenty-one years before her first attack, large arteries and veins formed suspension cables in some areas between the cortex and dura mater. Associated with these more or less external changes, we invariably have found an area of cortical cicatrization, thick, firm, grayish-yellow in color and easily recognizable in differentiation from the normal brain tissue. Often the center of this cone-shaped tissue was soft, gelatinous and cystic.

Such was the condition found in a patient who received a severe skull injury as a boy and who had his first convulsive seizure eight years later. After an interval of another thirteen years during which convulsions occurred at irregular intervals, he developed weakness in the right face, arm and leg and presented himself for treatment. At operation the cortical scar was blocked out and removed *en masse*. In spite of a period of freedom from attacks for eight months, the convulsions returned. He was placed on carefully controlled therapy but died in status epileptocus.

The appearance of the brain after surgical removal of the originally damaged tissue approximates very closely the condition found at operation except for the smaller size of the scar.

In 2 cases we removed a foreign body in the center of the fibrotic tissue; in one instance a screw and in another a bullet, with complete relief of the seizures. In 2 patients, rib transplantation was performed to prevent recurrence of the adhesion between the scalp and dura mater. One of these latter patients is now free from convulsions though it is necessary for him to take 30 grains of bromides daily in order to keep his blood bromine content at 125 mg., which for him is the non-convulsant level. In one instance we removed a large calcified mass from within the cortex which had evidently developed from an area of hemorrhage, and the patient has had no more attacks though he has never received anti-convulsant therapy.

In evaluating the results following operation we find that there was no improvement in one patient, another died in status epilepticus after an eight month period of freedom; another patient had no attacks in nine years though it must be noted that there was a free interval of six years before operation. In one patient the character

of the attacks changed. For six years he had no major convulsive attacks but "dreamy states" occurred about once every six to eight weeks. Other patients have been free from attacks for a year or less without medication.

Our results in those patients with large cortical defects, following injury at birth or accompanied by congenital defects in the skull, have been equivocal. The most satisfactory experience occurred in a boy aged ten years, who had a left hemiparesis and homonymous



FIG 175.—Encephalogram showing unilateral dilatation of the lateral ventricle of a patient with hemiplegia and Jacksonian convulsions, a result of *encephalitis* in infancy.

hemianopsia. His Jacksonian convulsions had been present since his birth by a prolonged instrumental delivery. A ventriculographic study showed an enormous, somewhat localized dilatation of the body of the right lateral cerebral ventricle. The roof of this portion of the ventricle consisted of a thin, vascular, transparent, gelatinous structure. When it was opened, the ependymal lined cavity was filled with clear cerebrospinal fluid in which a moderately hypertrophied choroid plexus floated The choroid plexus was removed from the lateral ventricle and only the dura mater remained to cover the *cortical* defect. Surprisingly, the hemiparesis disappeared, and for seventeen years he has had no attacks.

In contrast, are the remaining 4 patients in this group all of whom had a localized area of cystic degeneration of the cortex and in 3 of whom there were overlying defects in the skull. Resection of the area of obvious gross pathological change in no way modified the course of the convulsive seizures. In each of these patients encephalographic studies clearly demonstrated the mechanical defect in the ventricular system and subarachnoid spaces.

There were 5 other patients all of whom had localized Jacksonian attacks, with definite motor weakness and reflex changes following their convulsions. None of these patients gave a history of trauma at or following birth, and there were no evidences of congenital defects, but in each there was a clear history of an infectious illness followed by the apparent residue of an acute or subacute encephalitis.

Here again the dubious character of the clinical results of operative interference is obvious. In one patient whose right-sided convulsions began at the age of thirteen years, in whom evidences of paresis were present and who was in a status epilepticus, surgical exposure of the brain revealed generalized increase in the cortical vascularity and evident thickening of the arachnoid. The operation was followed by an entire freedom from seizures for seven years. *It is difficult to attribute such a clinical result to a surgical procedure* which amounted only to inspection of the brain. It is still harder to explain why the patient had a recurrence of similar attacks.

In 3 other patients with a similar clinical study and with almost identical findings at operation, no such false hopes of a cure were raised. Their attacks continued unchanged in frequency or character in comparison with their preoperative condition.

In contrast, the remaining patient in this group presented encephalographic evidence of a dilated ventricle, its roof pulled toward the scarred and gliotic cortex which had the consistency of a piece of gristle. One might justifiably believe that resection of this area, with demonstrable return of the ventricular system to the midline, would be followed by improvement, but such was not the case. Within a year the ventricular roof and lateral wall were again pulled outward by a contracting cicatrix, and at no time were the convulsions materially altered.

Electroencephalographic tracings taken directly from the exposed cerebral cortex have shown that the marginal cortex about the area of the lesion is the source of convulsive discharges. It is not the gross lesion, *but on the contrary the actual focus of neuronal hyperirritability*, in which electroencephalographic spikes and sharp waves originate, which causes the attacks. As yet the question cannot be answered if the epileptogenic focus is stable or if, with the passage of time, it does not set up other epileptogenic foci

Patients who are suitable for surgical removal of the epileptogenic focus must have some evidence of localization of the origin of the seizure. This evidence may be apparent from the history of the attack, that is, the characteristic pattern of events which occur; neurological findings, electroencephalographic tracings, pneumoencephalograms, or arteriograms. Once the firing point of the attack has been determined, the only serious obstacle to its surgical removal is the physiological and psychological importance of the cerebral area in which it happens to be located.

As yet, the long term results of the surgical treatment of convulsive seizures are far from satisfactory. Even when obvious cortical-meningeal scars, atrophic cortical areas or similar macroscopic lesions are identified electrically as firing points, the results indicate only about 20 per cent of the patients can be expected to be free of their convulsive attacks. This percentage is still lower in those patients in whom the cortex may appear to be grossly normal and in which identification of the trigger point depends mainly upon electrocorticographic and electrostimulative methods. Failure, of course, may occur when the patient has two or more firing points, only one of which is identified at the operating table

In 1941, Penfield and Erickson[1] reported upon 165 patients operated upon in a ten-year period with a mortality of 4.2 per cent. In the group of patients with excision of a meningocerebral scar, 22.5 per cent were completely free from attacks; 65.5 per cent were at least 50 per cent improved, 11 per cent were unchanged; and 2 per cent were worse. In the group of patients with a cerebral cicatrix, 19 per cent were completely free from attacks; 49 per cent were improved, 30 per cent were unchanged; and 2 per cent were worse.

Like other fields in neurological surgery, this problem remains for solution It cannot be dismissed as completely unfruitful, but neither should operations be undertaken except under conditions which approximate those of the experimental physiological laboratory as closely as is possible Certain it is that freedom from attacks in patients three, five or even seven years after operation, cannot be presented as evidence of the complete cure of their attacks, but it is to be hoped that the accumulation of accurately reported data may eventually lead to the establishment of surgical indications and may provide a valuable link in the solution of an intricate neurological problem ages old.

[1] Penfield, W and Erickson, T. C. "Epilepsy and Cerebral Localization," Volume 12 p 623, Charles C Thomas, Springfield, Illinois, 1941.

CHAPTER XIII

THE SURGICAL TREATMENT OF INTRACRANIAL VASCULAR LESIONS

"An aneurism is the dilatation of an artery full of spiritous blood."—
JEAN FERNEL, 1581.

INTRACRANIAL vascular lesions may be classified into 3 groups; (1) *arterial aneurysms* such as those which occur most commonly at various locations on the internal carotid artery, or its branches; (2) *arteriovenous fistulæ*, for example, the fistula which occurs between the internal carotid artery and the cavernous venous sinus, and (3) *angiomas*, which may consist of the abnormal dilatation and looping of a single vessel, or a cluster of dilated vessels which may be regarded as a tumor.

Aneurysm.—An aneurysm of an intracranial artery is produced by the force of the stream of arterial blood upon the congenital faulty wall of the vessel. This usually consists of a partial to complete loss of the muscularis, elastica and intima of the vessel. This defect may eventually take the form of a tiny out-pouching, or a pedunculated mass, and it may occur along the course of the vertebral, basilar or internal carotid arteries, either extracerebrally at the base of the brain or within the substance of the brain. These developmental defects in the wall of the vessel are left during the complicated stages of resolution of the intracranial vascular tree during which period budding, growth, resorption and re-arrangement occurs until the final vessel pattern is established. It was once believed that lues and direct trauma were the most common etiological factors but this is not true.

A single aneurysm of considerable size, or even multiple aneurysms, may be found upon the vessels which constitute the Circle of Willis as incidental autopsy findings and the patient may never have shown a single clinical sign of such a lesion. Usually, an aneurysm produces its first symptoms only after it has leaked blood. This may be so slight as to produce only stiffness of the neck and transient weakness in the extraocular muscles innervated by the third, fourth and sixth cranial nerves. Pupillary inequality, ptosis and diplopia may be accompanied by irritation of the root, ganglion or branches of the trigeminal nerve resulting in pain, paresthesia or actual loss of sensation in the face. In a similar manner, aneurysms along the course of

a vertebral or the basilar artery may slowly destroy the last four cranial nerves to produce weakness and anesthesia of the soft palate, hoarseness, difficulty in swallowing and atrophy of the corresponding half of the tongue. Large aneurysms alongside the clinoid processes of the sella turcica may produce erosion of the bone, as they may also occur at the foramen magnum.



FIG 176 —Traumatic intracranial arteriovenous aneurysm. Photographs above show the marked exophthalmos and dilatation of the scleral vessels. Photographs below show the recession of the exophthalmos and the scleral vessel dilatation following ligation of the left common carotid artery.

When an intracranial aneurysm suddenly ruptures with the outpouring of a quantity of blood, the results may be a dramatic fatality. Or, the patient may have prodromal nausea and headache and then suddenly, have a severe intracranial pain and lose consciousness for a few hours or for several days. Convulsive seizures, either focal or generalized, may occur. Usually, there are no signs which indicate the location of the lesion during the immediate post-hemorrhagic period even when small neurological evidence is carefully and meticu-



Fig. 177 Traumatic intracranial arteriovenous aneurysm showing marked edema of conjunctiva and proptosis.

lously sought. It may not be until consciousness is regained that lateralization of the lesion can be made. At any time after the initial dramatic onset, a lumbar spinal puncture will reveal grossly, bloody cerebrospinal fluid unless the aneurysm lies within the cerebral substance or within the cavernous portion of the carotid artery, in which case, the blood never reaches the subarachnoid space. The bleeding usually ceases spontaneously after the first rupture if the patient is kept quietly in bed, and when he regains consciousness a few lateralizing signs may make their appearance. Chances for a spontaneous cessation of the bleeding are much less with the second or third hemorrhage. For this reason, diagnosis and treatment should be given as soon as possible after the initial episode of bleeding.

Bleeding from an aneurysm into the cerebral substance, or into the cavernous sinus, produces immediate and unmistakable evidence of localization and lateralization of the hemorrhage. If there is a massive hemorrhage from an aneurysm in any location into the ventricles, the patient dies quickly from respiratory and circulatory collapse.

Since the proposal of cerebral arterial visualization was made by Moniz[1] in 1927, this procedure has been used more and more extensively to visualize the vascular system of the brain by roentgenological examination during the introduction of radiopaque material into the arterial blood stream. Injection of the radiopaque substance into the carotid system will fill the ipsilateral cerebral vessels and usually those of the anterior two-thirds of the Circle of Willis. In about twenty per cent of the injections the posterior cerebral vessels will also fill. By appropriate compression of the contralateral carotid artery during injection, bilateral filling of the anterior and middle cerebral vessels may be demonstrated. The posterior cerebral vessels and those of the posterior fossa and vertebral system may be demonstrated by either direct or indirect vertebral artery injection.

Various techniques may be used for the procedure of arteriography and none of them are without danger, as is true in any procedure which has to do with a seriously ill patient. Diodrast has almost completely replaced thorotrast as a contrast medium although even the former in a 35 per cent aqueous solution is far from ideal insofar as it may cause reactions in an individual sensitive to the drug or in one whose renal status is poor. The problem of testing patient for sensitivity to the drug is controversial since the usual skin and conjunctival tests are notoriously inaccurate and intravenous testing has been known to cause death. In most patients a direct percutaneous injection of the common carotid artery may be done or, the injection may be made directly into the internal carotid artery which has been exposed through a small incision in the neck. A large

[1] Moniz, E., "L'Angiographie Cerebrale," Masson et Cie, Paris, 1934.

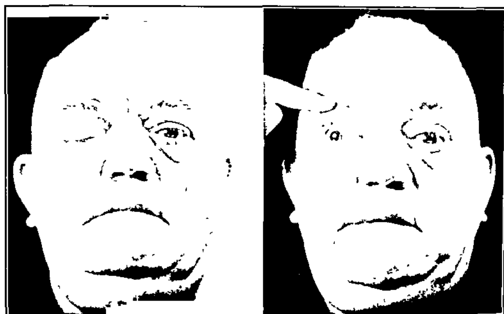


FIG. 178 —Intracranial aneurysm which produced an opthalmoplegia.

FIG. 179 — Angiogram which demonstrates an intracranial aneurysm.

hematoma may rarely form in the neck as a result of leakage at the puncture site when the percutaneous method is used and when pressure has not been maintained over the puncture site after withdrawal of the needle. Although the internal jugular vein lies over the common carotid artery very often and may be perforated by the needle in the percutaneous method, there have been no reports of the later development of an arterio-venous fistula. The vertebral artery may be injected percutaneously at its location opposite the fifth or sixth vertebræ or it may be filled by injection into the right common carotid artery with compression above the point of injection and over the brachial artery thus causing a retrograde flow of the diodrast through the innominate and subclavian arteries to fill the vertebral system.

Roentgenograms of the skull are usually taken at 2, 4, and 6 or 7 seconds after the start of the injection and many ingenious methods that have been devised are in use to obtain several films, or a set of serial films, during the injection. Thus, the arterial, capillary and venous phases of cerebral circulation may be shown.

Regardless of the technique, there must be close cooperation between the surgeon and the roentgenologist to obtain successful arteriograms. Ideally, the procedure is performed under local procaine infiltration, but if that cannot be used then penthothal anesthesia with an endotracheal tube in place is satisfactory. The patient may experience a marked hot, burning sensation over the side of the face and eye during the injection of the drug and should be forewarned so that his co-operation will be sustained. Flushing of the face and conjunctival injection may also occur.

The most conservative method of treating an intracranial aneurysm is that of ligation of the common carotid artery in the neck under local anesthesia in order to test the effect of occlusion of the vessel before it is finally ligated. The intima of the vessel should not be injured beyond the point of ligation, or an arteriosclerotic patch must not be fractured or dislodged within the vessel at the site of ligation; therefore, gentleness must be exercised during the operation. Ligation of the common carotid, just below its bifurcation, will not shut off all [illegible] Supplied by collate[illegible] opposite side, the internal and external maxillary, lingual and superior thyroid arteries will provide a stream of blood into the external carotid and then into the internal carotid artery. Consequently, for complete obstruction of all blood flow through the ipsilateral carotid system to the hemisphere, the internal carotid artery would have to be ligated at a point between its origin from the common carotid and its entrance into the base of the skull. So far as the neurological effects of obstruc-

tion of the blood supply to one cerebral hemisphere are concerned ligation of the internal carotid artery is a more serious and drastic procedure.

In an occasional patient, a transient hemiplegia will follow common carotid ligation, particularly if the patient is old and arteriosclerotic, or the blood pressure is low. However, this usually disappears after a few days. In our experience, this has happened six times in a series of 110 carotid ligations. There is no dependable rule to

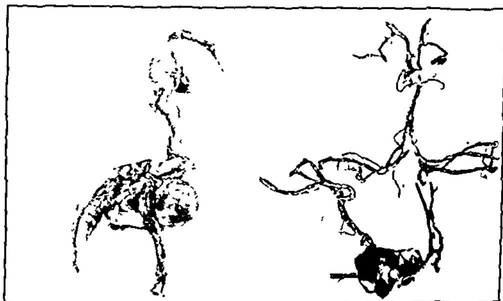


FIG. 180 —Dissected autopsy specimen of an intracranial aneurysm.

follow in judging from an arteriogram whether or not carotid ligation will be followed by undesirable neurological effects. However, if the anterior cerebral artery does not fill, then a block in the carotid system between the point of injection and the junction of the anterior communicating and the ipsilateral anterior cerebral artery may be assumed to be present. If the patient is without hemiplegia on the opposite side at the time, the anterior cerebral artery probably receives an adequate supply from the opposite side through the anterior communicating artery. Fortunately, in most patients who do not have an aneurysm of the anterior communicating artery, or the proximal portion of the anterior cerebral artery, an adequate blood supply will come to the hemisphere through the anterior communicating vessel.

It has been said that occlusion of the common carotid artery is of little value in the treatment of aneurysms well beyond the origin of the internal carotid artery. However, Bakay and Sweet[1] have

[1] Bakay, L. and Sweet, W. H. "Cervical and Intracranial Intraarterial Pressures with and without Vascular Occlusion." Surg. Gynec. and Obs., 95, 67, 1952

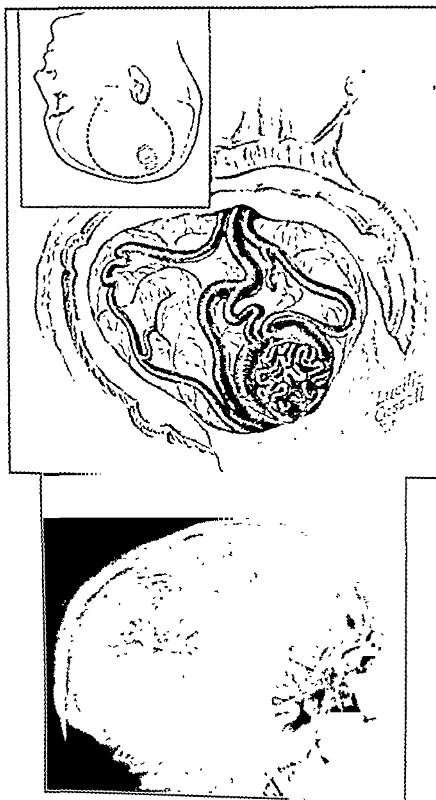


Fig. 181.—Artist's drawing of a cerebral arterio-venous fistula exposed at operation with angiogram of a similar case.

shown from direct measurements that there is a decided fall in the pressure even in small arteries on the superolateral surface of the cerebral hemisphere when the ipsilateral common carotid artery is closed. These findings give physiological proof for the good clinical results which follow this operation.

Upon occasion a pedunculated aneurysm may be trapped between silver clips if a main artery is not involved, or if its base is sufficiently distinct, it may be clipped so as to leave the lumen of the vessel patent. However, the vessel walls near an aneurysm are soft and fragile and not ideally suited for the placement of ligatures. Moreover, at the site of old or recent hemorrhages, all the surrounding structures are stained and it is difficult to elevate the soft overlying brain without fresh hemorrhage. The mortality and postoperative neurological residuals are much greater than in the more conservative ligation of the common carotid artery which prevents the direct forceful thrust of the blood stream upon the weakened arterial wall.

If the aneurysm lies intracerebrally, it may be attacked through an osteoplastic craniotomy, the cortex incised, the clot evacuated and the vessel ligated on either side of the lesion.

A pedunculated aneurysm on the vertebral artery may be ligated at its base but to attempt to trap such an aneurysm on either side may result in disabling neurological defects, or death. Ligation of the basilar artery will result in partial decerebration and death.

Arterio-Venous Fistulæ.—A pathological connection between arterial and venous systems in the form of one or more wide thin-walled vessels is characteristic of these vascular formations. These replace the normal capillary system, if the fistula be congenital in origin, within the region of the lesion. The most striking example of an intracranial arteriovenous fistula of traumatic origin is that which not uncommonly occurs between the internal carotid artery and the venous cavernous sinus which surrounds it.

The characteristic and diagnostic symptom of these fistulæ is a bruit, which may be accompanied by a thrill, and is best heard over the point of communication but may be transmitted distinctly for some distance. This bruit may be recognized by the patient, but often is not, and requires careful, repeated examinations, sometimes with amplification apparatus, to be detected by the examiner. It is produced by the passage of a forceful current of arterial blood through a small opening into a vessel with a lower pressure. The veins distal to the aneurysm dilate since no capillary bed exists between the artery and vein, so that large pulsating masses of thin-walled vessels develop. The proximal arteries dilate in compensation, and this extends even to the heart. The ophthalmic vein, imbedded in loose areolar tissue and with normal thin vein walls, dilates from

the arterial pressure which is forced within it. This produces a pulsating exophthalmos which is characteristic of a fistula between the internal carotid artery and cavernous sinus. The veins of the sclera become enormously dilated, and the conjunctiva of the eye becomes markedly edematous.

Basal skull fractures and non-fatal gunshot wounds of the skull are the two most common causes of an intracranial arterio-venous fistula. In one of our cases, the patient was struck in the left parieto-occipital region by a brick. Six months later he noticed a protrusion of the eyeball, and in the following month he had a pounding, whistling noise in his left ear. This was more marked when he lay on his left side. Exertion, such as lifting a weight, made his eye become more prominent. Diplopia finally developed, which brought him to the hospital. The left external rectus muscle was paralyzed and the exophthalmometer reading was 35 millimeters on the left as compared to 28 millimeters on the right. A pronounced bruit was heard over the entire anterior half of the skull. It could be stopped completely by digital compression over the left common carotid artery, which was ligated with complete disappearance of his symptoms and recession of his eye. We have found it necessary, as have other surgeons, to ligate the internal carotid artery to obtain permanent relief and possibly because the common carotid was ligated first and the internal carotid later, no neurological defects have resulted. Or, a collateral circulation has been established sufficiently before operation, so that occlusion of the internal carotid artery was accomplished safely.

Arterio-venous fistulæ which occur on the surface of the cerebral hemispheres are often congenital in origin, although they may, of course, develop as the result of direct trauma to the skull. Subarachnoid hemorrhages, Jacksonian convulsive seizures, transient attacks of hemiparesis, hemaniopsia, headaches and a bruit audible to the patient and examiner, or to neither, are the usual clinical symptoms. Dilated vessel grooves in the skull or small, fine calcific deposits may give roentgenological evidence on plain films but the positive diagnosis can be made by angiography. The angiogram shows a well-defined wedge-shaped formation of closely placed, small, tortuous vessels, which cross each other irregularly so that the whole has the appearance of a ball of twine, or a tangle of earthworms. Leading to this are one or more arteries, dilated and tortuous, which as a rule differ distinctly from normal arteries. The draining veins are also greatly dilated and fill the contrast medium earlier than normally because of the rapid circulation.

For the surgeon it is of great importance to know about all the feeding vessels leading to the fistulous mass if it is to be attacked

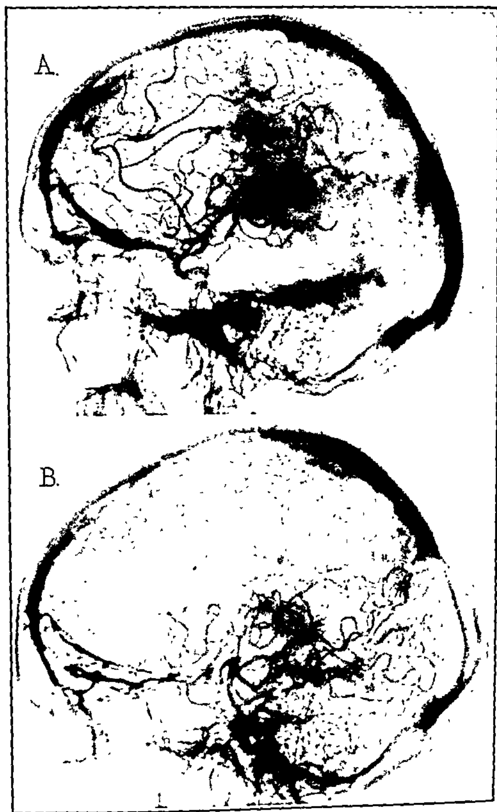


FIG. 182 (*A*) Arteriogram following right common carotid artery injection (*B*) Same patient following right common carotid artery injection with pressure on carotid artery above the site of injection and pressure occluding right brachial artery This procedure results in visualization of the vertebral artery

surgically by excision, or ligation of vessels. The internal carotid artery on the opposite side must be visualized if the aneurysm lies near the midline. If it is located basally, it may receive blood supply from the posterior cerebral artery also and this vessel cannot be filled by carotid injection. It should be injected through the vertebral artery. The results of excision of these fistulæ, or ligation of the principal vessels, is accompanied by considerable danger as far as residual neurological defects are concerned. In several instances we have performed a carotid artery ligation in the neck, which has stopped the bruit, allowed a considerable interval to elapse, and subsequently again visualized the mass by angiography before attacking it directly.

Angiomas.—These congenital malformations have received many names, such as, angiosis, angioma arteriale racemosum, angioma venosum racemosum, varix racemosum, cirsoid angioma, and angioma cavernosum. They may consist of a single arterial dilatation, or a single venous dilatation; multiple arterial or venous dilatations or a combination of these. Finally, there may be a port wine nevus of the face with cerebral cortical telangectasis. In these malformations, the cerebral lesion may be a pie-shaped tumor mass of combined arteries and veins, or there may be a discrete area covered by a mass of very fine vessels appearing like a large strawberry. Angiography is now established as the diagnostic method to be used in these patients, who may show fine calcium deposits scattered within the tumor mass on the plain roentgen-ray films In the past roentgen-ray therapy has been used in an attempt to shrink the vessels which constitute the tumor mass but it is very doubtful that it has been helpful. Certainly, an attempt at the surgical removal of these lesions may be a harrowing experience.

CHAPTER XIV

THE SURGICAL TREATMENT OF THE PSYCHOSES

In 1936, Moniz[1] described an operation which involved destruction of the anterior portion of the frontal lobe for the treatment of the psychoses. He introduced the terms, "pre-frontal leukotomy" and "psycho-surgery." There are many objections expressed to this method of treating psychotic patients because the early results were not uniform and because there was considerable doubt that psychotic patients could be benefited by a destruction of cerebral tissue. Since that time several different surgical procedures have been proposed but the common feature of all is the interruption of frontal lobe fibers which lead to the thalamus. As a result, degenerative changes occur in the medial dorsal nucleus of the thalamus. In the beginning the major problem was to weigh the benefits from the operation against the defects, and this has not yet been completely solved.

It should be realized that destructive lesions of the frontal lobe, or elsewhere in the brain, will lead to some neurological or psychological defect, but at the same time it may produce results in the psychotic patient which are distinctly beneficial from the viewpoint of the patient's total behavior. To strike the balance between these liabilities and assets, requires good psychiatric judgement based upon careful observation and conservative methods of treatment of the patient. However, in general and based upon the collective experience gained from careful clinical studies, it is agreed that tension, apprehension, fear, concern, worry and agitation are reduced or abolished. Emotional outbursts, excitement, aggressive behavior and exhibitions of rage tend to diminish and disappear after a latent period of a few weeks or months following operation.[2] Paranoid ideas lessen or, at least, do not lead to unbridled expressions. Obsessive thinking becomes less distracting and compulsive actions do not occur. Hallucinations of long standing do not disappear entirely but may lessen or may produce a minor response.

Since many of the patients operated upon have been ill for many years, often it is difficult to decide how much of a post-operative defect is due to the destructive brain operation and how much is the result of the condition which existed when the operation was per-

[1] Moniz, E "Tentatives Operatoires Dans le Traitement de Certaines Psychoses," Masson Cie, Paris, 1936.

[2] Solomon H C "Prefrontal Leukotomy, an Evaluation," Jour. Am Med Assn., 140, 1079, 1949.

formed. Tests of general intelligence have not been found to be particularly helpful; impairment is slight in tests of concrete and abstract behavior; personality tests (Rorschach) show a tendency for the patient to perseverate and give stereotyped and conventional responses, but color tests for emotional reactivity show greater freedom and less rigidity in responses. Certain reactions following operation may be considered as defects. A carefree attitude, with euphoria, may be excessive for the best type personal relations; apathy, lack of interest, facetiousness, profanity and distractability may be found immediately after operation, but there is a definite tendency for these reactions to improve. Obviously, the greatest concern is over the loss of judgement, future planning, discrimination, laudable ambition and creative imagination.

From a summary of observations and impressions regarding the efficacy of the different surgical techniques employed in the treatment of mental diseases at present,[1] certain opinions have been expressed regarding the results in particular mental states. Some have stated that patients with *manic-depressive psychoses* and *involutional melancholia* who have failed to benefit from the best environmental conditions, psycho-therapy and convulsive shock therapy, are proper candidates for operation. Others say that this group responds well to electro-convulsive therapy and are disinclined to perform an operation which leaves lasting scars on the personality. Obviously, the decision to forsake more conservative treatment and employ surgical procedure requires the best psychiatric judgement. It is agreed that patients with *obsessive-compulsive neurosis* of long standing and with symptoms which totally incapacitate them, who have had the benefit of the best and most intensive psycho-therapy and who, it has been concluded will not undergo remission, may be operated upon as a logical step in treatment.

The largest group of patients in any psychiatric hospital consists of those suffering from *dementia praecox* or *schizophrenia*. This group is the most challenging one in which to evaluate the results of operation and constitutes the largest group of patients which have

[1] Kalinowsky, L. D. "Principles Governing the Selection of Cases for Psycho-Surgery"

Freeman, Walter "Lobotomy—A Comparison of Pre-Frontal Lobotomy with Transorbital Lobotomy"

Solomon, H C "Psychiatric Evaluation of Results Following Unilateral Prefrontal Lobotomy Under Direct Vision"

Hoch, P. H. "Evaluation of the Results of Topectomy Operations"

Wilk, E. K "Selective Cortical Undercutting-Results in New Method of Fractional Lobotomy"

Spiegel, E A, and Freed, H "Evaluation of the Results of [illegible] and Other Surgical Procedures for the Relief of Psychoses"

Surg., Gynec, and Obs, 92, 601, 1[illegible]

been operated upon. It is also true that these patients are the ones in whom other methods of treatment are so inadequate. Many of these patients recover or show remissions in the first year of their illness. Kalinowsky starts all patients with acute schizophrenia on 20 electro-convulsive treatments, giving three treatments a week. If the response is good, but relapse occurs, another series of ten treatments is added. A third attempt with intense or "confusional" electro-convulsive therapy which produces 2 to 4 convulsions every day may then be instituted. If all of this fails, insulin coma treatment is begun and if this, in turn, is followed by a relapse then operation is advised. He believes that the hebephrenics are poor risks for psycho-surgery because they present emotional flattening and disorganization of thought. It is stated that the delusions, hallucinations and aggressive tendencies which schizophrenics have toward themselves and others can be modified by operation. It is agreed that the results of operation are best if the patients have not become deteriorated and this is usually the case in those patients who develop the disease after the age of forty and the group of patients who have been regarded and treated as neurotics for many years but who have a bizarre train of symptoms and somatic delusions. It appears, therefore, that in schizophrenic patients there is a critical point during the course of the illness before which the operation is worth while and after which it is inadvisable. Again, careful psychiatric judgement is necessary.

Solomon has stated that patients whose psychosis is associated with organic disease of the brain seldom benefit from lobotomy and that there is no evidence to show that the operation will benefit the patient with a psychopathic personality.

There is a group of patients who have suffered from *intractable pain* for many years, who have become addicted to drugs and in whom less mutilating procedures have proven inadequate. These patients have benefited, in our experience, from either unilateral or bilateral lobotomy, as the case has required.

At present there are at least six different types of operation used for the treatment of mental disorders. *Prefrontal lobotomy* is performed through burr holes placed in the coronal suture of the skull. The white matter of both frontal lobes is severed usually in the plane of the coronal suture and sphenoidal ridge. Freeman and Watts have said that incisions placed too far anteriorly do not relieve the mental disorder, while those placed too far posteriorly are crippling to the personality. Freeman developed a procedure, proposed first by Fianberti, of *transorbital lobotomy*, in which the severance of the frontal fibers is made blindly and reports an operative mortality of less than 2 per cent. Poppen has described a simple

surgical technique in which the fibers are divided under direct vision by the electro-coagulation suction unit. Scoville has described *selective cortical undercutting*, a method of functionally and anatomically isolating various areas of the frontal cortex with preservation of its own and the adjacent blood supply, making a line of cleavage with a brain spatula and fine suction tube at the relatively avascular junction of gray and white matter, thus interrupting long and short association fibers to the thalamus. This method selects the superior cerebral surface, chiefly areas 9 and 10 of Brodmann, the orbital surfaces of the frontal lobe and the medial surface, including the anterior cingulate gyrus both above and below the corpus callossum. Pool has employed a fractional ablation of the frontal lobe cortex (*topectomy*) in which the resection concerns only the gray matter separating the white matter almost entirely, and in which 25 to 35 grams of tissue are removed on each side. Wycis and Spiegel have performed *thalamotomies*, using a modification of the Horsely-Clark stereotaxic instrument for human patients. They have produced bilateral lesions in the dorsal medial thalamic nuclei, the anterior thalamic nuclei and the lateral part of the posterior hypothalamus.

As experience increases in the surgical treatment of patients with mental disorders, there is slowly appearing some degree of unanimity of opinion among psychiatrists who have evaluated the results of these various surgical procedures. Solomon states that the experience of the Boston Psychopathic Hospital with the use of lobotomy operations in patients with a chronic psychosis has produced relatively satisfactory results. Real improvement in behavior has resulted, making the life of the patient more comfortable. Life outside an institution has been possible in 40 per cent of the patients and about one-half of those have become relatively self-supporting. While in some instances, there has been no evidence of any demonstrable loss, he believes that an assessment of effect following operation is difficult to make and in this respect psychological tests have not been a great help.

Hoch believes that markedly aggressive patients respond better to lobotomy than to topectomy, but otherwise the results have been similar. It is his impression that marked alterations in personality, such as apathy, complacency, and emotional dulling do not occur as frequently and persistently following topectomy as they do after lobotomy

Wilk has said that complete lobotomy produces too much blunting of the personality to be used except in severely deteriorated psychotic patients. Selective cortical undercutting, he believes, indicates that there is little specificity of the frontal lobes in their effect on the

psychoses but definite specificity in their effect on personality. The psychoses are favorably affected by quantitative isolation of any area of the frontal lobes. Only fractional lobotomy should be used in patients suffering from mood disorders, neurosis, or pain. Undercutting of the orbital surface is regarded as the ideal operation for psychoneurosis and milder mood disturbances, because of the absence post-operatively of personality changes. Undercutting of the superior surface of the cortex, or of the orbital surface, is recommended for the schizophrenic and severe affective psychotic patients

Although there has not been ample opportunity to compare thalamotomy with the various other surgical procedures, Spiegel and Freed believe that the stereotaxic method of thalamotomy, requiring careful application of the apparatus for pre-operative roentgen-ray studies as well as for the operation, should be studied more extensively because of the fact that this method may produce beneficial effects with minimal neurological and psychiatric residual symptoms

It is correct to say that experience and careful evaluation of the results by psychiatrists should result in the choice of patients for whom the operation of dividing association and projection fibers from the frontal lobe will be of real benefit.

CHAPTER XV

THE SURGICAL TREATMENT OF ABNORMAL INVOLUNTARY MOVEMENTS

THERE are certain diseases of the central nervous system which are characterized by abnormal involuntary movements and alterations in muscle tone. These are often referred to as extrapyramidal system diseases. Among the most common are choreo-athetosis, Parkinson's disease, post-encephalitic Parkinsonism, hemiballismus, dystonia musculorum deformans, spasmotic torticollis, and hepato-lenticular degeneration.

The abnormal involuntary movements which characterize these diseases may be grouped into those bizarre, unpredictable and non-repetitive, quick or slow movements which more commonly involve the peripheral part of an extremity rather than the proximal portion. The movements are purposeless and do not have even the appearance of a purposeful movement. In some diseases, although the extremities are involved the trunk undergoes torsion movements of great magnitude also. The term *choreo-athetosis* has been applied to these movements.

These involuntary movements may follow infections of the central nervous system, such as infantile encephalitis or influenzal encephalitis Developmental deficiencies, or degenerative diseases, of the basal ganglia may result in athetosis, Huntington's chorea, paralysis agitans or hepato-lenticular disease. Vascular lesions which destroy parts of the basal ganglia may be responsible for these disabling, hyperkinetic movements which are almost unceasing during the waking state, but which stop during sleep.

Hemiballismus is less commonly encountered but the movements are vigorous, extensive, rapidly executed, without pattern or purpose and involve only one side of the body.

Other types of involuntary movements are the *tremors* which occur at rest, static tremors, and action tremors. These are involuntary, regular and rhythmic movements which occur over and over again at a definite rate. A tremor at rest occurs in a supported extremity which is not taking part in voluntary movements. A good example of this type of tremor is that which occurs in *paralysis agitans* or in *post-encephalitic Parkinsonism*. A static tremor occurs in muscles which are holding a part of the body motionless. Action tremors occur in muscles which are engaged in moving a part of the

body and the latter two may be placed together under the common term, intention tremors.

The character of these abnormal involuntary movements depends entirely upon the part of the central nervous system involved. *Paralysis agitans*, with its tremor at rest, results from a destructive lesions of the globus pallidus, substantia nigra or both. *Choreo-athetoid* movements most commonly follow a lesion of the putamen and caudate nuclei of the basal ganglia, while *hemiballismus* is due to a lesion of the subthalamic body of Luys. *Intention tremors* usually follow a lesion of the nerve fibers which arise from the dentate nuclei and pass through the thalamus and thence to the pre-central motor cortex. Obviously, these destructive lesions in the portions of the central nervous system named cannot produce the involuntary movements. Bucy[1] has explained this paradox on sound anatomical and physiological facts by pointing out that two large fiber systems originate from Areas 4 and 6 of the pre-central motor cortex. One consists of the large pyramidal tract which extends from the cortex to the spinal cord. The second consists of extra-pyramidal tracts which go to subcortical areas and are relayed to other parts of the central nervous system, including the spinal cord There is physiological evidence which suggests that the subcortical areas suppress or inhibit motor cortex activity. Therefore, he states, the involuntary movements so characteristic of these diseases in the basal ganglia are the result of release of control over the two large pyramidal tract systems. There is evidence that Area 4 is most concerned with tremors at rest and intention tremors, while Areas 4 and 6 are involved in choreo-athetosis.

It would appear most logical then in attempting to treat these diseases surgically to remove all or portions of Areas 4 and 6 in the pre-central motor cortex, to divide the motor projection fibers in the internal capsule or in the anterolateral portions of the medulla or spinal cord, or to place lesions in the thalamus which would interrupt one of the relay stations for these fibers.

Bucy has reported upon the extensive subpial removal of the pre-central motor cortex to abolish choreo-athetoid movements In fact, a part of the frontal gyri lying anteriorly is also removed. Only a partial paralysis results from this procedure because of innervation from remaining portions of the pre-central motor cortex on the same side and from the opposite cortical area. There is less disability from the paralysis in the leg because bilateral representation is greater than in the upper extremity and because the spastic nature of the paralysis causes less disability to the leg as a supporting structure.

[1] Bucy, P C "Surgical Relief of Tremor at Rest," Ann Surg, 132, 933 1945 "Cortical Extirpation in the Treatment of Involuntary Movements," Assn Research Nerv and Mental Diseases, 21, 551, 1940

If the patient's disease is progressive, or if it is bilateral, as is usually the case in choreo-athetosis and dystonia musculorum deformans, the operation is contraindicated. Removal of Area 4 only may be performed for the relief of the tremor of paralysis agitans. Hemiparesis results which the patient must accept for the relief of tremor. Tremors are rarely associated with paralysis in the affected extremity, as is choreo-athetosis, and therefore, this neurological residual defect is a major factor in the treatment of a progressive disease in which the muscular rigidity, disabling posture and gait, remain unchanged.

Browder[1] has sectioned the fibers which pass through the anterior limb of the internal capsule for Parkinsonism. Gross movements of the paralyzed extremity begin to return within two weeks after operation and improve to the point where the upper extremity is useful. However, he points out that unless a slight hemiparesis endures, the tremor will return.

Meyers[2] reports that 66 per cent of his patients with Parkinsonism were improved following section of the anterior limb of the internal capsule, or pallidofugal section. While there are fewer residual disabilities with these operations then with cortical extirpation, the surgical risk is definitely greater. He also states that pallidofugal section is not applicable to patients with *spasmotic torticollis*, for which the most satisfactory operation to date is that proposed by McKenzie of selective, bilateral section of the anterior spinal roots of cervical 1, 2 and 3 segments and the spinal accessory nerve.

Spiegel, Wycis and Freed have produced electrolytic lesions of the dorso-medial thalamic nuclei for the relief of involuntary movements in choreo-athetosis and have made lesions in the globus pallidus or substantia nigra to relieve the choreic movements in Huntington's chorea on the theory that lesions of these basal ganglia are characterized clinically by hypokinesis and such lesions than should be useful in hyperkinetic diseases. This would not appear to be compatible with Bucy's explanation for the release of hyperkinetic movements by lesions of the basal ganglia, except that globus pallidus lesions in disease produce only tremors and not the purposeless, gross, rapidly executed movements of choreo-athetosis.

It may be said that all operations thus far used for the relief of the abnormal involuntary movements of the extra-pyramidal diseases are experimental in nature. All are unsatisfactory in one form or another [illegible]
routinel[illegible]
which sh[illegible] understanding a group of diseases for which there is no other therapeutic hope at the moment.

[1] Browder, J.: "Section of the Fibers of the Anterior Limb of the Internal Capsule for Parkinsonism," Amer. Jour. Surg., 75, 264, 1948.

[2] Meyers, R.: "Experiments in the Therapy of Certain Extrapyramidal Diseases A Current Evaluation," Ejnar Munksgaard, Copenhagen, 1951.

CHAPTER XVI

CONGENITAL AND ACQUIRED ANOMALIES OF THE NEURAL AXIS

"That child has water on the brain, says the layman; that child is hydrocephalic, gravely says the physician, repeating literally by a Greek word what the layman says in his own language. But what is this water? Where does it come from? That is what the doctor should try to discover."—FRANCOIS MAGENDIE, 1842.

HYDROCEPHALUS

BEFORE Magendie drew attention to the physiological importance of the fluid which surrounds the central nervous system, the paper on "Dropsy in the Brain" written by Robert Whytt was the authoritative source to which all writers turned. Whytt gave an excellent description of a condition now recognized as tuberculous meningitis, and in his paper said, "the symptoms of no distemper resemble those of water on the brain so much as those which arise from worms in the stomach." The not uncommon modern practice of regarding the vomiting produced by the hydrocephalus which accompanies a midline cerebellar tumor as a symptom of a gastro-intestinal disorder does not allow us to criticize Whytt's statement too severely.

Early in his surgical career Cushing was impressed by the importance of the cerebrospinal fluid because of the considerable number of infants with hydrocephalus brought to him by desperate parents. Further, as he has so well stated, "the success or failure of many of his (the neurosurgeon's) undertakings depends more upon his familiarity with the fluid circulation and its possibilities as a complicating factor in his procedures, than upon any other one thing."

Much of what we now know of hydrocephalus has come from Cushing's interest in the matter of what he has termed "the third circulation" and the stimulus he transmitted to his associates. It was noted that in many of the hydrocephalic infants the intraventricular and intraspinal pressures were equal and were affected equally by straining or crying. It was also observed that although large amounts of fluid could be removed from the ventricles, so that the skull had the appearance of a bag of bones, the fluid reaccumulated within twenty-four hours. But there were other cases of obvious obstruction to the circulation of the cerebrospinal fluid by tumors, which showed only a small amount of fluid in the spinal sac.

Thus, from rather simple clinical observations, the conception of two types of etiological factors in hydrocephalus gradually developed. The one factor, that of obstruction, which prevented the fluid from circulating freely through the ventricles to the subarachnoid spaces, and the second, that of a developmental fault in the apparatus of outflow for the fluid from the meningeal spaces, or its occlusion by disease.

Dandy and Blackfan[1] produced an experimental hydrocephalus by occlusion of the aqueduct of Sylvius which caused the ventricular system cephalad to the obstruction to dilate. Later, two more observations, crucial to the study of hydrocephalus, were made by these same men. They set up an inflammatory reaction encircling the cerebral peduncles at the level of the tentorium and again found enlargement of the ventricles. This experiment added the significant fact that after escaping from the ventricles by the foramina of Luschka and Magendie, the fluid passes upward in the arachnoidea through the incisura of the tentorium to circulate over the surface of the cerebral hemispheres. The second experiment was suggested by Cushing's observation of fluid exuding from a choroid plexus exposed during an intracranial operation. In the laboratory Dandy was able to occlude one foramen of Monro and produce a unilateral hydrocephalus, but if at the same time the choroid plexus was removed, the ventricle remained collapsed.

Thus, it became obvious that hydrocephalus would result from an obstruction at any point in the pathway of circulation for the fluid from its origin in the choroid plexus to the meningeal spaces. Such a "non-communicating" hydrocephalus, commonly found in space-occupying lesions, can also be produced by pre- or postnatal inflammatory lesions. The point of obstruction can be easily determined by the introduction of a dye, such as indigo-carmine, into one ventricle with its subsequent recovery from the opposite ventricle and by lumbar puncture; or, by direct roentgen-ray visualization of the ventricular system by the injection of air.

However, this type of internal hydrocephalus is a problem usually secondary to the presence of an intracranial tumor. It is the "communicating," or "essential" hydrocephalus which affords an even more difficult therapeutic problem. Since, in a "communicating" hydrocephalus there would appear to be no obstruction to the flow of fluid, the fault must lie within the apparatus for absorption.

By injecting potassium ferrocyanide and iron-ammonium citrate into the subarachnoid space, Weed found that the Prussian-blue granules were precipitated in and passed through the mesothelial

[1] Dandy, W. E., and Blackfan, K. D: An Experimental and Clinical Study of Internal Hydrocephalus, Jour. Am. Med. Assn., 61, 2216, 1913.

cells which cap the arachnoid villi. The latter project in large numbers through the dura mater into the cortical and basilar venous sinuses. These observations confirmed the original theories of Key and Retzius regarding the absorptive function of the Pacchionian granulations, which are only pathological enlargements of a few of the multiple villi which exist. The processes of the manufacture, circulation and now the absorption of the fluid seemed clear. (Fig. 183.)

It is not difficult to realize that these many microscopic villi may very easily become occluded by inflammatory products or by blood in the meningeal spaces, thus reducing the absorptive powers of the *villi through which the fluid normally escapes into the venous sinuses.* Later, Weed produced a chronic hydrocephalus by injecting lampblack into the subarachnoid space, thus occluding the arachnoidal villi.



Fig. 183.—Infant with hydrocephalus of the communicating type.

In all of the children we have seen with hydrocephalus, which was obviously present at birth and may therefore be called congenital, ventriculography and dye injections have proved it to be of the "communicating" type; that is, the lesion affected the absorptive and not the circulating mechanism. The "non-communicating" group of cases were without exception proved to be due to space-occupying lesions. Russell's[1] excellent monograph on the pathology of hydrocephalus is the result of a comprehensive study of this condition Maldevelopments, inflammations, dural sinus thrombosis and thrombo-phlebitis, neoplasms and gliosis of the aqueduct, a condition not well understood, are the common pathological findings.

Maldevelopments.—Errors in development are frequent causes of hydrocephalus which result in such an enlargement of the head that delivery is impossible In other children, the hydrocephalus may not be evident until shortly *after birth; in still others,* the degree of the hydrocephalus may never become great enough to produce neurological symptoms Developmental defects are usually found at

[1] Russell Dorothy S "Observations on the Pathology of Hydrocephalus." Medical Research Council Special Report Series, 265, His Majesty's Stationary Office, 1949

the anatomical sites at which the cerebrospinal fluid pathway is constricted normally, *i.e.*, the foramina of Monro, the aqueduct of Sylvius or the foramina of Luschka and Magendie.

Simple stenosis and atresia, which implies complete non-patency, are common malformations of the aqueduct of Sylvius which produce congenital hydrocephalus. Russell has never encountered a complete absence of the lumen, but on the contrary has found "forking" of the aqueduct which she states is a more accurate description of the morphological abnormality.

Neuroglial septa in the aqueduct and at the foramen of Magendi may occur but precise microscopical evidence is lacking to prove that these are developmental errors.

The most commonly recognized cause of hydrocephalus is spina bifida, which may be divided into: (1) *meningo-myelocele* in which the nervous tissue of the spinal cord is spread out over the wall of the sac; (2) *meningocele*, in which the meninges and nerve roots are the only portions of the spinal cord in the sac, and (3) *spina bifida occulta*, which is characterized by a defect in the vertebral laminæ without any protrusion of the soft tissues. A tuft of hair, a dimpled area in the skin, a reddish-purple discoloration of the skin or a small fatty tumor over the site of the bony defect may be the only external evidence of this developmental defect.

A malformation which consists of a tongue-like prolongation of the cerebellum caudally and which overlaps and is found to be a greatly elongated medulla oblongata is known as the *Arnold-Chiari malformation.* Russell and Donald found it present in 10 consecutive cases of spina bifida of the meningo-myelocele type. There are many variations in the actual anatomical relations but in every case the spinal cord appears to be displaced caudally into the spinal canal. The association of a communicating hydrocephalus with this severe form of spina bifida is well established, and is explained by displacement of the fourth ventricle foramina into the spinal canal. This produces an obstruction to the spread of the cerebrospinal fluid into the cranial cavity due to plugging of the foramen magnum.

It has been commonly observed that surgical excision of the sac in spina bifida is often followed by hydrocephalus or exaggeration of an existing hydrocephalus. Penfield and Cone demonstrated that the walls of the sac had the capacity to absorb spinal fluid and that it acted as the means for passage of the fluid into the blood stream. If then, the Arnold-Chiari malformation blocks the return of cerebrospinal fluid into the cranial cavity and the sac absorbs fluid, removal of the latter will precipitate or exaggerate the hydrocephalus. Others have attributed the development of hydrocephalus in cases of

spina bifida to stenosis of the aqueduct caused by traction upon and elongation of the mid-brain.[1]

Greater variations in the occurrence of these anatomical malformations in the less severe degrees of spina bifida, such as meningocele and occulta, occur in Russell's opinion but have been described infrequently. Hydrocephalus may co-exist with spina bifida occulta and the patient may show no evidence of spinal cord involvement; on the contrary, the occurrence of spina bifida occulta in the cervical region with syringomyelia has been well documented.

Platybasia is a deformity of the skull in which the basal angle formed by the base of the sphenoid bone and the clivus, normally 130 to 140 degrees, is increased. A variety of disturbances may arise from the pressure of the bone upon the brain stem and nerve roots but the medulla and cerebellar tonsils may also be pushed into the upper cervical spinal canal. Under these circumstances and with a narrowed foramen magnum, hydrocephalus may occur as the result of a mechanism similar to that in the Arnold-Chiari malformation.

Gliosis of the aqueduct of Sylvius has an obscure etiology and must be distinguished from simple stenosis, forking of the aqueduct and obstruction of the lumen due to a fibrillary astrocytoma of the quadrigeminal plate. Some have regarded it as a malformation and Spiller stated that it was the result of an excessive closing-in of the lumen such as occurs in most spinal cords in the thoracico-lumbar segments Small groups of ependymal cells may be found in the glial tissue around the aqueduct in normal specimens. Russell points out, however, that there is an excessive amount of fibrillary glia in the region of the aqueduct in cases of gliosis, which makes a neoplastic theory attractive. Finally, the possibility of an inflammatory process is present but there is little real evidence in its favor.

Inflammations.—Reference has been made to the experimental production of hydrocephalus by the injection of particulate matter. Similarly, the local breakdown of cells within the ventricles, the extravasation of red blood cells, or abnormal metabolic products may produce a low-grade inflammatory reaction with fibrosis of the leptomeninges and a resulting obstruction in the cerebrospinal pathway, particularly in the subarachnoid basilar cistern.

In the trauma of birth, severe hemorrhages from tears of the tentorium and the great vein of Galen are familiar, but lesser non-fatal hemorrhages may occur which are the forerunners of obstructive lesions in the subarachnoid spaces In some instances, small intracranial hemorrhages may occur in utero before any mechanical trauma is possible.

[1] Lichenstein, B W "Distant Neuro-anatomic Complications of Spina Bifida (Spinal Dysrathism)," Arch Neurol Psychiat, 47, 195, 1942

Certain non-bacterial infections with organisms such as *Monilia*, *Torula* and *Toxoplasma* may produce a meningo-encephalitis which seals off the foramina of the fourth ventricle and results in hydrocephalus.

Ventricular dilatation may follow progressively during the course of a severe acute purulent meningitis but the more chronic bacterial inflammations produce sequelæ due to obstructions in the leptomeninges. There is evidence that infections of the meninges in the newborn infant are not uncommon and, of course, may go undetected until hydrocephalus develops. The arachnoid of the basal cisterns appears opaque especially over the cisterna magna and the foramina of Luschka. While the history of infection may be obtained, bacteriological proof of its presence may not be forthcoming. Infection with a Flexner bacillus or B. Coli are probably not uncommon factors in producing an illness in the newborn, the pattern of which is extremely difficult to recognize.

Thrombosis and Thrombo-phlebitis of the Dural Sinuses.—The cerebrospinal fluid may be produced adequately and may circulate perfectly but cannot be absorbed properly into the blood stream through the arachnoidal villi which project into the dural venous sinuses. The disease process may be a very slow and spreading one which is not immediately productive of ventricular dilatation. While experimental efforts to reproduce this mechanism of hydrocephalus have failed, there is clinical evidence which supports this etiological factor plus the fact that the thrombophlebitis may be localized, and an extensive thrombus may not be demonstrable, and yet a hydrocephalus be the result.

Neoplasms.—Intracranial tumors so located as to obstruct the cerebrospinal fluid pathway produce a secondary hydrocephalus. This becomes quickly obvious in children whose skull sutures have not closed and affords them early relief from the effects of increasing intracranial pressure. The percussion note of the hydrocephalic child is characteristic and pathognomonic. Tumors of the fourth ventricle, colloid cysts of the third ventricle, tumors of the midbrain and cerebral hemispheres, and intraventricular tumors all may produce an enormous dilatation of the ventricular system cephalad to the point of obstruction to the flow of cerebrospinal fluid. Tumors of the third ventricle and midbrain are often surgically inaccessible and in these patients short-circuiting of the cerebrospinal fluid from the lateral ventricle, through a catheter which lies beneath the scalp and ends in the cisterna magna (ventriculo-cisternostomy) provides relief from symptoms which is remarkable in these slow-growing tumors. Likewise, diffuse tumors of the leptomeninges at the base of the brain, diffuse melanosis, primary sarcomatosis and

metastatic tumors, produce an obstruction in the fluid pathway which results in ventricular dilatation.

Surgical Treatment.—Many of the early surgical attempts to relieve hydrocephalus were based upon an incomplete and inaccurate knowledge of all of the factors involved in the production, circulation, and absorption of the cerebrospinal fluid. Under the impression that the fluid normally escaped into extracranial and extraspinal lymphatics, attempts were made to drain the subarachnoid lumbar fluid into the retroperitoneal tissues by a trephine opening in the body of the fifth lumbar vertebræ. These efforts, as had those which attempted to establish permanent drainage into the subaponeurotic layer of the scalp, met with disappointing results. The operation devised by McClure, which involved the transplantation of a vein between the subdural space and a vessel in the neck and Cushing's attempt to place a silver tube from the third ventricle through the corpus callosum directly into the longitudinal sinus were both followed by inconclusive clinical results. Wegefarth also suggested that hydrocephalus might be treated by making openings directly through the longitudinal sinus into the leptomeninges with the expectation that each opening would be filled by arachnoid and that these artificially produced villi might furnish a lack in absorptive function.

A surgical attack upon the point of obstruction in a "non-communicating" hydrocephalus is definitely indicated when this has been demonstrated. Removal of a neoplasm, ventriculo-cisternostomy, opening arachnoidal adhesions which occlude the cisterna magna or lateral cisterns, are logical surgical procedures. If there be any capacity for the absorption of fluid in a "communicating" hydrocephalus, removal of the choroid plexus is the most logical step and the one most likely to succeed in spite of its natural hazards

The first attempt to remove a choroid plexus for hydrocephalus with which we are familiar was made by Dr. V L. Lespinasse of Chicago In 1910, he introduced a small cystoscope into the ventricle and fulgurated the plexus bilaterally in two infants. One of his patients died post-operatively, and the second lived five years. The method was presented before a local medical society and was not otherwise recorded. Other endoscopic methods have been developed for coagulating the choroid plexus with more modern instruments Our experiences with ventriculoscopes has been unfortunate as compared to direct visualization of the plexus by opening the ventricle This may serve in selected cases to arrest the process in about 50 per cent of the patients

At present the surgical outlook for patients with hydrocephalus is not as entirely hopeless as it once was, but as Cushing said, "though we may not appear to be much nearer to a cure for congenital hydrocephalus, in quest of which we started out, we have at least learned many things on the way which are essentially of practical therapeutic value."

SPINA BIFIDA

We have referred to spina bifida and its relationship to hydrocephalus in the preceding pages but these conditions are encountered so commonly as to justify further consideration.

The story of spina bifida is an old one, and because it is one of the most impressive of all developmental defects, it attracted the interest of the superstitious physicians of old. Spina bifida is the result of an incomplete union of one or more laminæ of the vertebral column, occasionally of the bodies, with or without protrusion of the spinal cord or its membranes. The condition is such a striking error in development that it is necessary to remember some few embryological facts to understand completely the condition.

The neural tube becomes separated from the epiderm by mesoblastic tissue which in turn is responsible for the outer meninges, vertebræ, muscles, and associated ligaments During the second fetal month, six cartilaginous centers appear and then join to form the cartilaginous vertebræ. In the fourth month, the laminæ begin to unite to form the spinous processes and this extends caudally and cephalad from the dorsal region.

Just what mechanism results in the development of spina bifida is not known satisfactorily though during the course of time many theories have been advanced.

In 1816, Béclard suggested that as a result of torsion of the umbilical cord, accumulations of fluid developed in certain locations and gave rise to spina bifida, hydrocephalus, and related anomalies. The St. Hilaires, father and son, minimized the effect of torsion of the cord and attributed more importance to the persistence of fluid which occurs in developing embryonic nerve tissue. Later, Daresti stressed the importance of adherence of the amnion to the surface of the developing embryo.

The Report of the London Clinical Society in 1885 stated that the mesoblast failed to separate the neural tube from the epiderm. Von Recklinghausen suggested that the origin of the condition resided in the notochord.

More modern embryologists are in agreement that spina bifida, like most congenital malformations, is the result of a deficiency in the structure of the chorionic villi, cells and blood-vessels. Such an explanation places the blame on the decidua rather than upon the ovum, but whether the defect lies primarily in mesoblast or epiblast remains unsettled.

Pathologically and clinically, spina bifida may be classified into two types: complete and partial.

Complete spina bifida is incompatible with life; and therefore, the three varieties of partial spina bifida, *meningocele*, *myelo-meningocele* and *spina bifida occulta*, are more commonly observed.

Spina bifida occulta may occur with little or no symptomatic evidence of its presence. A small fold or dimple, a tuft of hair, or a small soft mass in the midline of the back may be the only external evidence of an imperfection in the spinous processes or laminæ of one or more vertebræ. (Fig. 184) This bony defect is most commonly found upon roentgen-ray examination of the spinal column, particularly in the lumbo-sacral region. We have observed several patients

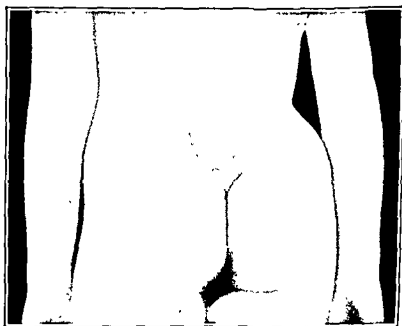


Fig 184 —Spina bifida occulta.

with a spina bifida occulta in the cervical vertebræ, who had the associated symptoms of a progressing syringomyelia. The only surface evidence of this malformation was a small dimple in the skin, directly in the midline, out of which a tuft of long hair grew.

The spinal cord and membranes are normal in structure and position, but a band of fibrous tissue bridges the space between the deep surface of the skin and the coverings of the spinal cord. As body growth increases, it is possible though it is not common for increasing tension upon this band to produce spinal cord or root symptoms.

midline of the back is filled with cerebrospinal fluid and [illegible] light. It may be covered entirely by normal skin, but [illegible] the skin is thin and partially ulcerated over the upper half [illegible] tumor. The mass is painless unless an attempt is made to re[illegible] contents into the spinal canal by pressure. As a rule, no [illegible] weakness or sensory disturbances exist below the lesion.

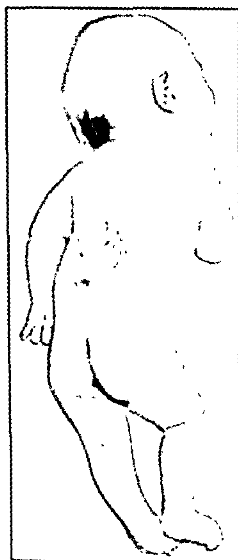


Fig. 185.—Infant with myelo-meningocele

Anatomically, the spinal cord is normal though a nerve root may be herniated into the sac. The dentate ligaments are usually well formed, but the vertebral spinous processes and laminæ are represented by small stump-like bony projections.

In our own experience this type of spina bifida is comparatively favorable for treatment but is met with less often than the more

serious *myelo-meningocele*, in which spinal cord and nerve roots are herniated into the meningeal sac. The clinical appearance is similar to a meningocele except that it is more formidable and repulsive. The fundus of the cystic, elongated mass is commonly bluish in color, and over this area is a very thin layer of epidermis which seems as if it might give way at any moment.

When the sac is examined carefully, a segment of the spinal cord will be found to have remained in its embryonic state as a part of the neural plate. The sac is usually multilocular, and its cavity is crossed by a group of nerve roots which arise from the elementary neural plate and pass forward to enter the intervertebral foramina.

Whereas, it is unusual to find evidence of neurological symptoms in a simple meningocele, motor, sensory and trophic disturbances are common in myelo-meningocele and vary with the extent of the lesion. It is not uncommon to find harelip, cleft-palate, clubbed feet, or extroversion of the urinary bladder as associated embryological malformations.

By far the largest number of patients with spina bifida we have seen have been those whose lower extremities were paralyzed and who had relaxed urinary and rectal sphincters, the result of a myelo-meningocele. It is in these patients that the problem of surgical indications and judgment play such an important rôle. It has been our own practice to reserve operation for those patients in whom there seems to be some opportunity of restoration of function. For example, the pure meningocele is the most suitable for operation. In about one-half of the patients with a myelo-meningocele have we obtained results which seemed to justify the operation. We have refused operation upon twice that number of patients because of ulceration of the hernial sac, leakage of cerebrospinal fluid, or massive paralysis. It is our practice to advise operation between the third and fifth months, or even later if the condition of the sac wall justifies delay. It is true that many of these infants have succumbed during the delay, but they have remained marasmic and have in each instance failed to present the slightest encouragement for surgery. Some surgeons make it a rule that extensive paralysis of the lower extremities, rectum and bladder are absolute contraindications to operation. Others state that over one-half of all of the cases of spina bifida are suitable for operation, but this has not been our experience The presence of hydrocephalus in an infant with spina bifida is generally accepted as a surgical contraindication, and the development of hydrocephalus following an operation for repair has been a serious deterrent to a successful result (Fig 186.)

The principles underlying the plastic repair of the defect should include the preservation of the sac, which has been shown to have

the properties of a fluid-absorbing tissue.[1,2] After the sac is opened, evacuated of fluid and sutured again, it should be rolled up and placed beneath protective layers of muscle fascia. The flaps of muscle and fascia should remain attached at the edges of the defect and may be imbricated in layers to cover the defect.

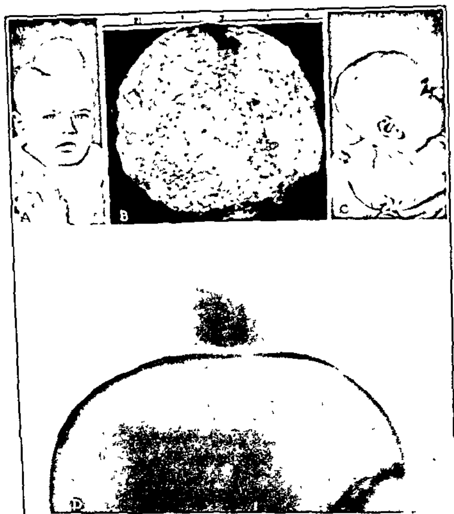


Fig. 186.—(*A*) Photograph of an infant, aged seven months, with a craniomeningocele which originated from the anterior fontanelle; (*B*) photograph of the meningocele which had a very small pedicle attachment; (*C*) photograph of the infant after operation; (*D*) roentgen-ray film showing the relation of the meningocele to the skull.

[1] Russell, D. S., and Donald, C.: Mechanism of Internal Hydrocephalus in Spina Bifida [illegible] ...o-meningo- ...he cervical ...This defor- ...blocks its ...from the fourth ventricle to the basilar cisterns. The meningocele sac therefore assumes the function of fluid absorption to replace in part the cerebral subarachnoid *spaces*.

[2] Penfield, W.: Hydrocephalus and Spina Bifida, Surg., Gynec. and Obst., 60, 363, 1935.

Our own group of cases is not large enough to answer the question of operative mortality and the more interesting and important ethical, social, and economic problem of the future of the patient so operated upon. Fraser's data[3] from the Edinburgh Sick Children's Hospital over the years from 1898 to 1923 give some fair conception of the situation. Of 191 patients, 130 were chosen as suitable for operation. Eighty-two were discharged from the hospital, an operative mortality of 37 per cent. In 1929, 46 patients were traced and 30 were found to be living, "but the greater proportion were crippled in body or in mind or in both" . . . These patients included all types of spina bifida, and it is fair to conclude that the unsuccessful and discouraging results were obtained in patients with myelomeningocele.

Ingraham and his associates have published an analytical survey of 462 cases of spina bifida and 84 cases of cranium bifidum observed at the Boston Children's Hospital during twenty years. It emphasizes the fact that associated anomalies such as hydrocephalus, club foot, bone and central nervous system maldevelopments are very common and in their opinion do not constitute a contraindication to surgical treatment if they are not progressive. The myelomeningoceles constitute the largest group. The various types of lesions are classified and their relation to sex, age, site and symptoms are presented in a manner that makes this contribution an authoritative source on this subject. These men found that 34 per cent of the patients with encephalocele and 30 per cent with spina bifida led a relatively normal life after operation.

As has been stated our most encouraging and successful results have been obtained in patients with a meningocele. We find it necessary to individualize each patient, as to the most favorable time for operation, taking into consideration the physical condition of the patient and the condition of the covering of the sac. In our own experience the development of hydrocephalus following repair of a spina bifida is less likely to occur when the operation can be delayed until the infant is three or more months of age. Perhaps the absorptive function of the sac becomes less important and that of the cerebral arachnoidal villi more securely established after that lapse of time

TERATOMAS AND TERATOID TUMORS

Spina bifida and meningocele formations of varying degrees usually accompany sacrococcygeal teratomas, which are large protuberant masses which deform the buttocks. These tumors may extend behind the sacrum to such an extent as to involve the bladder and rectosig-

[3] Fraser, John Spina Bifida, Edinburgh Med Jour., 36, 284, 1929.

moid portion of the colon and produce mechanical obstruction. In addition to this aspect of these tumors, they may be potentially malignant because of the epithelial elements within them. These tumors are large in proportion to the size of the patient and are usually extremely vascular so that their surgical removal may constitute a formidable procedure. It is necessary to obtain a firm closure of the dura and to preserve any portions of the cauda equina involved within them.

Intracranial and intraspinal teratoid tumors and teratomas do not have a constant position and they are not usually as highly differentiated as are the sacrococcygeal masses. Craniopharyngiomas and chordomas have been considered in the chapter on Intracranial Tumors. Ectodermal defects often overlie the teratoid tumors in the posterior fossa and spinal canal. In several of our patients a sinus in the skin which discharged thick, cheese-like material, was found overlying the lesion and in others a bifid defect in the spine was found on the roentgen-ray films.

INFANTILE HEMIPLEGIA

Cairns has drawn attention to the encouraging results which he has obtained by hemispherectomy in the treatment of infantile hemiplegia.[1] He has also classified these infants clinically by describing the onset of their hemiplegia: (1) the infant may be hemiplegic at or soon after a difficult birth, (2) the child may appear healthy at birth, but within the next four years has an illness characterized by convulsions, coma and fever, often diagnosed as encephalitis and, (3) there may be no previous acute illness, and convulsions and hemiplegia are the first symptoms.

At any age from two to twenty years, treatment is sought for these patients because of convulsions or mental abnormality. The hemiplegia is spastic and the hand and arm are more affected than is the leg but both extremities are shorter and smaller than the unaffected side, and flexion contractures of the wrist and fingers, and talapes equinus are present. Athetoid-like movements are commonly present in the upper extremity and involuntary relaxation of the hand or individual fingers is impossible. Cairns points out that sensation of the paretic side is impaired and often a homonymous hemianopsia is present

The cerebral hemisphere on the opposite side is small, its ventricle is dilated and porencephalic or subarachnoid cysts may be present

[1] Cairns, H. "Hemispherectomy in the Treatment of Infantile Hemipleg The Lancet, Vol. 2, 411–415, Sept. 8, 1951.

The ventricles are shifted toward the affected side and the skull is smaller on the affected side, as is the face.

Convulsions may not appear until several years after the hemiplegia and may be of any variety or frequency. These children are also often behavior problems in that attacks of screaming and temper tantrums may be the psychomotor equivalents of the convulsive seizures. As the years pass, it becomes increasingly evident that the child is slow to learn or previously acquired motor patterns and abilities may disappear.

In 1945, Krynauw operated on such a patient and in his efforts to remove all of those portions of the hemisphere which were easily stimulated electrically he eventually removed the entire cerebral hemisphere except the caudate nucleus, thalamus and perhaps the hippocampus. Following operation, his patient, a child of 9 years, ceased having convulsions, improved mentally so that she could attend an ordinary school, and there was no deterioration of the function of the affected extremity. Cairns operated on three such patients whom he followed for one year post-operatively. In all patients, the convulsions ceased; the hemiplegia was not worse but in fact was better because the spasticity was less and the mental state was dramatically improved.

Cairns believed that not all patients with infantile hemiplegia are suitable for hemispherectomy but it should be undertaken in those instances in which convulsions cannot be controlled by drugs and in which there is a deterioration in the patient's intellectual or emotional state. It is essential before operation to establish, by clinical examination and air studies, that one hemisphere is diseased and the opposite one is healthy. The many questions of cerebral physiology which these experiences has raised, such as the site to which control of voluntary movement of the contralateral limbs passes, and the reasons for the remarkable mental improvements shown, remain to be answered. Meanwhile, there appears to be an encouraging avenue of surgical approach for these unfortunate patients who, heretofore, have been the center of unhappiness for the entire family

INDEX

B

C

Q

R